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ON CEREBRAL FAT EMBOLISM: REPORT OF A CASE WITH RECOVERY.

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THOUGH slight fat embolism of the lungs is said to be in all probability very common after fractures, it is usually a matter of no practical importance, since the symptoms, if any, are so mild as to pass unnoticed. It is not always unimportant. Pulmonary fat embolism may cause serious symptoms. Fat embolism of the brain, however, is very serious and is usually a fatal condition, but fortunately it is rare, so rare indeed that even though the symptoms may be clamant, the diagnosis is liable to be missed through want of familiarity with the symptom complex.

It is remarkable that though the authors of students' text-books describe many things that very few hospital residents are likely to see, they give very scanty attention or none at all to the means of recognizing fat embolism, cerebral or otherwise. Here is all that the most widely used of all our students' text-books has to say about the symptoms of fat embolism:

> The pulmonary obstruction may, however, become so great as to lead to a fatal issue from dyspnœa; whilst if the cerebral vessels are blocked, syncope or even coma may be induced.

As regards cerebral fat embolism, readers of the cases that I shall be quoting in this paper will be able to judge for themselves whether the young house surgeon is given anything worth having when he is told that "if the cerebral vessels are blocked, syncope or even coma may be induced." As a matter of fact, I have been unable to find any record of syncope in any case anywhere. It would appear, therefore, that the information is not only meagre, but positively misleading. Our text-book says nothing about delirium and nothing about any petechial eruption. Under the circumstances hospital residents have ample excuse for knowing nothing about the condition and for missing it. That is why persons suffering from cerebral fat embolism, clamant as were their symptoms, have been (as I know) buried with their condition undiagnosed. On the other hand, as we shall see, equivocal cases occur which it is admittedly very difficult to diagnose from *delirium tremens*, surgical shock or other conditions.

So far as I know, I have had but one patient with cerebral fat embolism in the past six years. That was in November, 1919. I have waited for more, but they have not yet appeared. My patient was

very fortunate, because he survived. I give an account of his case later on, but I think it will be better to study the condition first through observations that have been made by others.

I have looked into the literature referring to the subject of fat embolism and studied a number of case records and other clinical and pathological information published in various journals in the past twenty years. There is much that is interesting, but of all that I have read, the most useful for present purposes, that is as regards cerebral cases, is a paper by Gauss.[1] I think readers will find that the most satisfactory method of projecting a picture of the condition before them is to give an abstract of Gauss's paper, transcribed from my own notebooks, as follows:

He quotes from a paper by Le Count and himself on fat embolism in Transactions, Chicago Pathological Society, 1915, IX., 251. This was a study of fourteen fatal cases. In eight *delirium tremens* had been diagnosed clinically, although a history of alcoholism had not been definitely established in each of the cases. Œdema of the brain was observed in seven, fat droplets in the blood were noted at the *post mortem* examination in six, petechiæ in the skin or organs in nine. In the lungs of all were large numbers of fat emboli and in half of them there were microscopical hæmorrhages. In the heart muscle, fat emboli were found in thirteen, microscopic hæmorrhages in twelve and fatty degeneration was seen in six. In the kidneys there were fat emboli in the whole fourteen, microscopical hæmorrhages were observed in ten and fatty degeneration in thirteen. In the liver there were fat emboli in six and in twelve there were venous engorgement and fatty infiltration. Clinically, all of them after a variable period of consciousness passed into a restless stage and in twelve there was delirium. The delirium in eleven of them was so violent that restraint was applied and eleven passed from delirium into coma. In all fourteen there was an increased respiration rate and considerable dyspnœa. In all cases the pulse became weak and rapid. Urine and fæces were passed involuntarily in twelve, in the other two no record of it was made. Thirteen came into hospital with normal or subnormal temperatures and in all it rose steadily to the time of death, the maximum temperatures averaging 40.6° C. (105.2° F.) for the fourteen. The bones fractured were the femur in four, the humerus in three, the tibia and fibula in five, the calcaneus in one and the pelvis in one. The ages of the patients were from thirty-five to ninety years. The period of time from injury to death varied from two to seventeen days, averaging six.

The following remarks may also be noted:

> The emboli are carried to the *vena cava*, thence to the right heart, which pumps them to the lungs, where they become lodged in the capillaries. After a variable period some of the emboli are forced through the capillaries and return to the heart and are then sent out again into the general circulation to reach the various tissues, where they again become lodged in the capillaries. Here also they are forced through after a variable period, but are replaced by new emboli which had been temporarily arrested in the pulmonary circulation. The fat is finally excreted, at least in part, by the kidneys. . . . .

As to the time of appearance of the secondary changes, especially the hæmorrhages, there are different opinions. Ribbert says they appear after the third day. Gröndahl found them after fifty hours; Warthin after twelve hours. Ribbert thinks that a third of the deaths in fat embolism are from the changes in the brain. Gröndahl puts the figure at a half. As pointed out by Benestadt[2] the changes in the brain are not necessarily fatal. He reports three cases in which the patients suffered from symptoms of cerebral fat embolism following fractures. All three patients passed through the first two stages and subsequently recovered. This apparently means that they exhibited restlessness and delirium, but did not pass into coma. Gröndahl is referred to as having divided cases into stages: (i.) Initial, before onset of symptoms; (ii.) the restless stage, in which the patients frequently become delirious; (iii.) the last or comatose stage.

In Le Count and Gauss's series, all patients suffered from definite cerebral symptoms. Five were brought to hospital in the restless stage and one of them was wildly delirious. In the cases of these five, a period of several hours had elapsed since the injury. The others suffered from no distress on admission, but became restless after from twelve to twenty-four hours and became gradually comatose in from twenty-four to seventy-two hours. In two the pupils were contracted on admission, but dilated before death. One suffered from ptosis of one lid after admission and one from strabismus.

The following is a report of one of Le Count and Gauss's fourteen cases:

> On October 4, 1909, a railroad fireman, aged thirty-five, was struck on the head by a low viaduct while on the tender. He was picked up unconscious and his leg was found to be doubled under him. He was brought to hospital several hours later and was by this time conscious. He had a superficial scalp wound over the left parietal region, but no fracture was recognizable there. There was also a fracture of the tibia at the junction of the middle and lower third and of the fibula a little higher. He lived four days. There were no cerebral symptoms on the first day, except that he appeared dull mentally. He slept nearly all night. On October 5 he awoke at 8 a.m. and complained of pain in the back. He became rather restless and morphine was administered, but the restlessness continued during the morning and afternoon. In the late afternoon he became drowsy, then stuporous and failed to respond to question, but he could be aroused temporarily by supra-orbital pressure. On October 6 he was in coma. The pupils were pin-point, the eyes turned upward and there was slight strabismus, with deviation to the left. Later in the day the pupils enlarged and the coma deepened, so that he could not be aroused by supra-orbital pressure. He continued thus till death on October 7 at 3 p.m. His respiration on admission was normal. On the morning of October 5 it became very irregular and by the afternoon it was Cheyne-Stokes, in which form it continued. On October 7 he developed singultus, his breathing became laboured and there was dulness and bronchial breathing over the lower lobe on the right side posteriorly with many coarse râles. The respiration rate reached 64 before death. The pulse was 74 on admission. It increased steadily, reaching 164 before death. On October 6 he became cyanotic and this was more marked next day. Blood

drawn contained fat droplets. He perspired profusely. Urine and fæces were passed involuntarily. Petechial eruption: Petechiæ were first noticed at 8 a.m. on October 5 in the scapular regions. They developed rapidly, breaking out in crops. On the morning of October 6 they were all over the trunk and by noon the neck was covered with them. The temperature was 97.8° F. on admission. It rose steadily and reached 106.2° F. before death.

A *post mortem* examination was made the morning following death. There was a comminuted fracture of the tibia, a fracture of the fibula and a small fracture of the roof of the orbit. Over the trunk, especially the upper part, there were innumerable petechiæ, in some places clustered, but over the upper part of the chest they were a centimetre apart. Petechiæ were numerous in the pericardium and in the lining of the right heart and they were observed in the testicles and in the gastric mucosa. In microscopic preparations fat emboli were found in the brain, lungs, myocardium, kidneys, supra-renals liver and testes. All sections of the lung contained emboli in large amounts. Scattered areas of lung tissue contained alveolar spaces filled with hæmorrhagic exudate. All sections of the kidney contained emboli, chiefly in the capillaries of the glomeruli. Nearly all the glomeruli contained some emboli and about a third of them were completely blocked up by them. Many *vasa efferentia* were embolized. There were small intertubular extravasations of blood. The liver contained emboli in small amounts. All sections of the heart contained emboli. Most sections of the supra-renals contained emboli. A few were seen in sections of the testes.

Brain: On sectioning, numerous punctate hæmorrhages were observed in the white matter throughout the entire brain. The whole brain was sectioned systematically and pieces were stained in various ways and examined. Besides the general œdema, there were noticed numerous areas of focal œdema of small size. In addition to the gross punctate hæmorrhages, there were countless thousands of hæmorrhages of microscopic size present in every section of every part. Similarly, there were fat emboli everywhere, seen in every section stained for them. The capillaries of the whole brain seem to have been embolized more or less. Besides the hæmorrhages and fat emboli, there were small microscopic areas described as "focal necrosis." There were, in addition, nerve-cell changes everywhere, apparently secondary to the embolization. In many parts there were areas also of round-cell infiltration. The whole brain was, therefore, studded with minute lesions which must have amounted in the aggregate to millions.

I have quoted the above case because its characteristics can be called typical. Above all, the petechial eruption should be noted as almost a pathognomonic sign, though it is not invariably present. It is a very remarkable phenomenon and when seen, it practically settles the diagnosis, that is, of course, when it is taken into consideration in relation to all the other circumstances of the case.

I append two more cases in which no petechial eruption occurred. These were reported by Godlee and Williams[8] as cases which "presented unusual symptoms which the *post mortem* examination proved to be associated with, if not due to, cerebral fat embolism." It is remarkable that both these patients were injured in the same railway accident, on an occasion in which nineteen were admitted to the hospital at once, with four deaths.

A.B., a male, aged twenty five, was admitted with a fracture at the junction of the upper and middle third of the left femur. He was brought in about an hour after the accident; he had not at any time lost consciousness and there was no evidence of injury to the head. At 2 p.m. he was given chloroform and the limb was put up. At 8 p.m. he became unconscious and could not be roused; the pulse was 130 and the rectal temperature 103° F. The breathing was Cheyne-Stokes in character. He had an extensor plantar reflex on the right side, but there were no other signs suggesting any localized injury to the brain. No urine could be drawn off. Lumbar puncture gave a clear fluid and there was no increase of pressure. There was some vomiting. The low tension of the pulse and its rapidity seemed against a diagnosis of cerebral compression and it was decided not to operate. At 12 midnight the periodic breathing had stopped, the respirations were shallow and there was some cyanosis. On December 6 at 11 a.m., lumbar puncture was again performed; the fluid again was clear with no increase of pressure. The pulse was very feeble and rapid. There was very little alteration in the patient's condition during the rest of the day. Towards midnight the temperature reached 105° F., it having risen steadily during the day. On December 7 the coma was less and the colour had become better; the extensor plantar reflex on the right side had given place to a flexor. The pulse improved in tension. The temperature fell during the day to 101.3° F. On the 8th the general condition seemed better. The patient presented interference. The pulse became slower (128) and its tension increased and the temperature sank to 100° F. On the 9th, in the early hours of the morning, the patient became very cyanosed and he died at 8.55 a.m. The temperature before death was 99° F. The urine was afterwards found to contain fat.

Post Mortem Examination: There was no evidence of injury to the skull. There was no œdema of the brain or flattening of the convolutions. On incising the brain every cut surface showed thickly studded punctate hæmorrhages. There was slight broncho-pneumonia. There was no obvious sign of disease in the heart, kidney and other viscera. The left femur was fractured at the junction of the upper and middle third, the ends were not in apposition and there was a large quantity of extravasated blood. There was no crushing of the bone, but on sawing the bone longitudinally the extravasated blood was seen to extend the whole length of the medullary cavity. No other injury could be discovered. Sections were made of the brain, lung, kidneys and heart; they were stained with Sudan III., with the object of determining whether and if so to what extent fat embolism had occurred, with the result that in the brain, besides the extravasation of blood around the vessels which was well seen, some of the capillaries were found to contain fat. In the lungs and heart the vessels were filled with fat as if they had been injected for a demonstration of the blood supply of the part. In the kidneys fat was also found, but limited to the capillaries of the glomeruli. If we had not been on the lookout for fat embolism there was nothing in the naked-eye appearance of the brain and other viscera which would have suggested its existence and it would have been concluded that the capillary hæmorrhages in the brain were the only discernible lesions which could have accounted for death.

C.D., a male, aged thirty, was admitted at 10.30 a.m. suffering from shock. There was a bruise on the right side of the head and the zygoma was fractured. Both femora were broken and there was fluid in both knees. The left foot was extensively lacerated. He was given chloroform, the wound was cleaned up and the thighs were put up in splints. At midnight he was very restless, but quite conscious; the pulse was 160 and of low tension. The temperature was 102° F. On December 6 at 3.30 a.m. he awoke, vomited and then became unconscious. Cheyne-Stokes breathing developed, but there were no localizing signs of cerebral injury. At 7 a.m. he was deeply comatose. The pulse was 160 and of low tension. Double extensor response was present. The temperature was 102° F. Lumbar puncture withdrew some faintly blood-stained fluid under moderate pressure and the withdrawal of the fluid stopped the (Cheyne-Stokes) periodicity of the respirations. At 11 a.m. the condition was unaltered. The pulse was still of low tension and the pupils were unequal and small, but as there was evidence of injury to the right side of the skull, it was decided to trephine over the right middle meningeal artery. This was done, but there

was no increase of intracranial tension and no blood beneath the dura. The patient gradually sank after the operation and died at 2.30 p.m., the gradual progress of the symptoms having been in no way affected by the operation. The urine was afterwards found to contain fat.

Post Mortem Examination: Both femora were fractured transversely about the middle of the shaft. On opening the knee joints they were found to contain liquid blood and floating upon the top of the blood was a considerable quantity of liquid fat, the double streams of black blood and pale yellow opaque fat issuing from the incisions presenting a most unusual appearance. The anterior and outer part of the right tibial articular surface showed fissures which did not, however, extend into the shaft. In the left knee joint the posterior crucial ligament was ruptured and the anterior partly torn away from the tibia. The whole surface of the brain was redder than normal owing to the injection of the vessels; otherwise there was nothing to remark externally. On section there were a few punctate hæmorrhages. The hinder end of the cribriform plate was fractured with comminution. There was a vertical fracture in the anterior part of the temporal fossa, whereby part of the malar bone, including the external angular process and the zygoma, were detached as a loose fragment. The lungs were somewhat hyperæmic and œdematous. There was no obvious lesion of the heart, kidneys or other viscera. The brain and lungs were hardened in formalin, microscopical sections cut and stained with Sudan III. They showed the capillaries to be blocked with fat; in the lung the smaller arterioles were also filled with fat.

In their commentary on these two cases Godlee and Williams say:

Within about twelve hours they both developed a comatose state more or less suddenly, without any previous, noticeable pulmonary distress, the temperature rose rapidly, the pulse was of low tension and rapid and there were no localizing signs in the nervous system pointing to a lesion of any particular part of the brain. Both might have been thought to be cases of cerebral compression if it had not been for the character of the pulse.

They go on to stress further the importance in a differential diagnosis of the character of the pulse. They emphasize the point that the characteristic pulse of compression was never observed.

The reader of these case reports will notice resemblances and differences between the one quoted from Le Count and Gauss's series and those reported by Godlee and Williams. In the first one there was a profuse eruption of petechiæ in the skin, an indication which, taken with the history and accompanying symptoms, can be regarded for practical purposes as pathognomonic. A diagnosis in such a case ought not to be missed. But the absence of this positive sign in Godlee and Williams's cases made it a matter for very careful differential diagnosis and without confirmation by *post mortem* investigation a certain amount of diffidence would very properly be felt. Godlee and Williams had a very strong suspicion, but it is evident that they had this diffidence, at any rate at first. They are greatly to be congratulated on their acuteness in discerning the probable nature of the conditions. I have asked myself if I would have done as well. I am not sure that I would.

I think that on the question of diagnosis special regard should be directed to the point that the coma does not come on till a period of hours or days has elapsed. A low tension pulse is not unusual in patients comatose from cerebral shock, but in such cases coma is precipitate on the accident. A delayed coma may be due to extra-dural hæmorrhage. This would cause a slowing of the pulse. It is then that the condition of the pulse would be of assistance in distinguishing between extra-dural hæmorrhage and cerebral fat embolism. I do not think the presence of fat in the blood or urine should be given too much importance. Pulmonary fat embolism to some degree is apparently a frequent occurrence in fractures. It would be associated with lipæmia and lipuria. But cerebral fat embolism is not common.

Having prepared the way thus far, I proceed now to report my own case.

J.M., aged thirty-three, a wharf labourer, was struck by a bag of sugar on the left leg on November 3, 1919. The tibia and fibula were fractured. He was admitted to the Sydney Hospital at 10.15 a.m.. The leg was put up in back and side splints. The X-ray report of same date was: "Fracture of both bones in middle third. There is fibular displacement of the lower tibial fragment." Nothing unusual occurred till the evening of the following day, when at 7 p.m.—thirty-five hours after admission—he complained of feeling ill. He was at once examined and the following report was entered on his case paper:

Face flushed. Respirations increased. Temperature 38.4° C. (101° F.). No dulness in front of chest. Harsh vesicular breath sounds, with generalized high-pitched rhonchi and fine to medium crepitations. Diagnosis, capillary bronchitis. Morphine gr. ¼ to be given.

This, therefore, was on November 4. The clinical report then goes on:

About midnight patient became comatose. Just prior to becoming so, he became slightly delirious, the temperature rising to 38.9° C. (102° F.). In the morning he was quite comatose. The pulse was full and the rate 106. The temperature was beginning to fall. The chest signs were absent, the breath sounds were now faint and vesicular. The right lower limb was slightly spastic, the knee jerk was slightly exaggerated and the plantar response was extensor. There were no signs of paralysis, the patient being able to move his limbs. The pupils were equal and reacted. There was incontinence of urine and fæces. The heart sounds were normal. There were no signs of thickened vessels. He was catheterized and about two fluid ounces of urine were withdrawn. This gave a heavy cloud of albumin and a slight reaction to the guiacum test. There was a remarkable petechial rash over the thorax. The spots were very tiny and did not disappear on pressure. Over the thorax the petechiæ were very numerous, but a few were present over the abdomen, arms and legs.

The above notes belong to November 5. For November 6 the report runs:

Patient conscious, very drowsy, memory blunted. He was unable to remember the day, etc.. Temperature down to normal. Reflexes all normal. Rash still present.

The temperature remained normal from this on and all symptoms quickly disappeared. A sample of blood drawn on November 6 for bacteriological examination gave no culture.

On November 20 the leg was immobilized in plaster of Paris and on November 25 he was allowed to go home with his leg in plaster.

It is so clear a case of cerebral fat embolism that to argue about the point would seem a waste of time. It is true that it was not proved by a *post mortem* examination, because the patient recovered, but that was really the only respect in which it did not conform to type. There was, of course, more than a mere fat embolism of the brain.

The record points very clearly to a severe preliminary disturbance in the lungs like a bronchopneumonia. Next day, although cerebral symptoms had appeared, the pulmonary disturbance had completely subsided, but the urine was loaded with albumin and appears to have contained a little blood. The interpretation can be given, I think, with full confidence, that the preliminary disturbance on the evening of November 4 was pulmonary fat embolism of a grave kind. I think we are equally safe in concluding that the albumin observed in the urine on November 5 was the result of fat embolism of the kidney.

Recovery after fat embolism of the brain is evidently very rare, for the condition is usually described in the books as a fatal one. Yet quite recently there appeared a report by R. H. Dahl,[4] in a Norwegian journal, of no less than three cases of fat embolism after fractures of the lower limbs, occurring within three months of each other, which were remarkable for the fact that though there were definite and severe cerebral symptoms (coma), complete recovery was effected. None of these patients had injuries to the head.

From personal inquiry I have become aware that there was a fatal case of cerebral fat embolism in the Sydney Hospital a few months ago. There was coma and the typical outbreak of petechiæ. The nature of the case was not recognized at the time, but the diagnosis is really beyond all doubt. The clinical notes are meagre and unsatisfactory. The patient was a woman with a fracture of the leg and not under my care.

Perhaps fat embolism may really play a larger part in the production of untoward results not only after accidents, but also after operations, than any of us have suspected. I do not mean the typical, easily recognized cerebral fat embolism, such as I have described in my patient, but others in which coma is not a feature. The three cases of recovery reported by Benestadt have already been referred to. In these, as in my case, there could be no proof by autopsy, because the patients did not die.

Worthy of careful attention is a paper published in 1917 by W. W. Bissell[5] from the Mayo Clinic under the title "Pulmonary Fat-Embolism—A Frequent Cause of Post-Operative Shock." He states that he had in the previous eight months observed six instances of fatal post-operative fat embolism in the necropsy service of the Mayo Clinic. Three of these followed breast amputations, one ventral herniotomy, one craniotomy for brain tumour and one laminectomy for spinal cord tumour. He gives the record of three of these in detail.

The first was an obese man, thirty-two years of age, operated on for ventral hernia on the afternoon of August 5, 1916, by a long transverse incision. He took the anæsthetic (ether) badly and was cyanotic for a considerable part of the time. At its close the pulse was of good quality and the rate 118 per minute. He improved during the next twelve hours. During the course of the next day the cyanosis persisted and there was considerable dyspnœa. The cyanosis and dyspnœa partially subsided by the end of the first twenty-four hours and the patient seemed to be on the road to recovery. During the course of the second day, however, the temperature rose gradually to 38.1° C. (100.5° F.) and in the evening he became mildly delirious. Delirium grew more intense and by the morning of the second day was associated with tremor and wild hallucinations of sight and hearing. The temperature rose gradually to 40.6° C. (105° F.) and the pulse rate to 140. Death occurred within forty-eight hours of the operation. During the second day the respiration rate rose rapidly, breathing was attended with great effort and there were signs of consolidation of the lobes of the lungs posteriorly. His history showed that he had been a bar-tender and up to three months before operation he had been a moderately heavy drinker. The clinical diagnosis of lobar pneumonia and *delirium tremens* was made.

At the autopsy, scattered over the skin of the abdomen and thorax there were a few (eleven in all) bright red petechial hæmorrhages about one millimetre in diameter. There were many bright red petechial hæmorrhages throughout the visceral and parietal peritoneum. The same phenomenon was observed in the pleura. There were coalescing petechial hæmorrhages in the lining of the stomach, duodenum, caecum and rectum. There were many small areas of consolidation in the lung parenchyma. Many fat droplets were visible on the surface of the blood escaping from the incised inferior *vena cava*. The same appearance was observed in the blood of the right and left auricles and in the blood escaping from the surfaces of lung sections, also in the blood of the dural sinuses of the brain. In microscopical preparations stained with Sudan III. fat emboli were found in the lungs, brain, kidneys, liver, heart muscle, spleen, adrenals and skin. A number of excellent photographs are given which display the microscopic appearances.

The second case was that of a moderately obese woman, aged forty-one, operated on on August 26, 1916, by enucleation of both breasts for fibro-cystic disease and by cholecystectomy for gall-bladder disease under the same ether anæsthesia. Recovery from the anæsthetic was normal. Within twenty-four hours the pulse rate rose to 120 and the temperature to 38.4° C. (101° F.). Some dyspnœa and slight cyanosis occurred and the patient became stuporous. During the third day the cyanosis increased and delirium followed. Before death the temperature rose to 39.6° C. (103.4° F.) and the pulse rate rose to 136. The clinical cause was given as surgical shock.

The *post mortem* examination disclosed moderate disseminated fat embolism. There were fat droplets in the fluid material from fresh cuts in the lung lobes, in the fluid material in either pleural cavity, in the tracheobronchial secretion, in the urine and in the blood clots of the right auricle. There were petechial hæmorrhages throughout the lining of the small bowel and part of the stomach, in the mucous lining of the pelvis of the left kidney, in the visceral pleura, in the peritoneum, the capsule of the liver and the visceral pericardium. No mention is made of the brain which perhaps was not examined. Photographs are given showing fat embolism in the lungs, liver and adrenals.

The third case was that of a moderately obese woman, fifty-six years of age, operated on September 9, 1916, for malignant disease of the breast by Halstead's method. She recovered promptly from the anæsthetic and the pulse was of good quality. Very soon, however, the pulse rate increased rapidly and the temperature fell below normal. The temperature continued subnormal and the pulse rapid, irregular and difficult to count. This condition continued for ten hours before death. There was no evidence of hæmorrhage. The clinical cause of death was given as surgical shock.

Post mortem there was found moderate disseminated pulmonary fat embolism, hypostatic hyperæmia and œdema of the lungs, petechial hæmorrhages in the mesentery and in the capsule of the spleen and pinhead-sized petechial hæmorrhages in the skin. As in the second case, no mention is made of the brain. Photographs are given showing the microscopical appearances in the fat embolized lungs.

Bissell then quotes a case reported by Roswell Park many years before in which coma, diagnosed

as due to fat embolism, followed operative removal of a cancerous breast.

While I think Bissell's idea that fat embolism or rather pulmonary fat embolism, is a "frequent" cause of post-operative shock, goes far beyond the logical value of his evidence—two cases—it is certainly remarkable that he should have discovered so many cases of post-operative fat embolism at the same hospital in the same year. We must admit that if such sequelæ to either accident or operation have occurred in one hospital, they may occur in another, whether diagnosed or not. We shall not diagnose them unless we have learned what to look for.

References.

(1) Harry Gauss: "Studies in Cerebral Fat-Embolism," *Archives of Internal Medicine*, July, 1916, Volume XVIII., page 76.

(2) Benestadt: *Deutsche Zeitschrift für Chirurgie*, 1911, Volume CXII., page 194.

(3) R. J. Godlee and G. E. O. Williams: "Notes on Some Victims of a Recent Railway Accident, With Special Reference to Cerebral Fat-Embolism," *The Lancet*, April 22, 1911, Volume I., page 1062.

(4) R. H. Dahl: Quoted in abstract in Epitome 231, *The British Medical Journal*, September 27, 1924, as from *Tidsskrift f. d. Norske Laege-Forening*, July 1, 1924, page 610.

(5) W. W. Bissell: "Pulmonary Fat-Embolism: A Frequent cause of Post-Operative Surgical Shock," *Surgery, Gynecology and Obstetrics*, July, 1917, Volume XXV., page 8.

THE FIRST SENSE IN MEMORY AND MEDICINE.

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How many of us realize the part that the sense of smell plays in our lives and how many students appreciate the value of smell differences in the diagnosis of disease?

In some animals at least after the primitive functions of touch and balance the sense of smell was early developed and played a greater part in the life of the individual than sight and hearing. This predominance still persists in many species, notably in the shark, some ants, dogs and many wild animals. In man the olfactory organs are probably undergoing a retrograde process. This is explained anatomically when we consider the square inch or so of smell-sensitive mucous membrane of man compared with the many square feet possessed by the shark.

With the evolution of the race this sense is losing its importance in the chase, in self defence and in the search for a mate, though the last-named function is fostered by the resourceful "flapper" in ways too well known to mention.

Let us dismiss at once the joys of the table and the saloon by reminding ourselves that most tastes, delicious and otherwise, really appeal to the olfactory and not the gustatory sense. With many of us memory has its strongest appeal in this elusive sense. A breath of salt-laden air from the sea and we are carried far away to childhood's days of rock ledges and tumbling breakers and we feel for a moment in the sultry city "the freshness of the early world." Again, the scent of a few crushed gum leaves transports us to eucalyptus gullies and sweet smelling camp-fires. No sight or sound can bring back to us so suddenly, so completely or so sweetly the perfect joys of super-days long past. Unlike most painful memories, unpleasant smells can transport us with speed of ether waves to scenes of horror and desolation. Pussy, poisoned and more than dead, recalls to us the poor, long-buried Turk who spoiled so many a simple repast. Boys playing with crackers, surround us in a flash with the stench of lyddite and the screech of high explosive shells. Apart from memory the sense of smell is always with us. For us Australians the desert of Mena, even the blue Ægean and the oft-sung Isles of Greece lost much of their charm in that their odours were foreign to us and we longed for the smell of the gum trees and the tang of the South Sea wind.

But I digress. Was it not medicine and diagnosis that I sought to discuss?

In examining a patient, Sir George Humphrey is quoted as having said: "Eyes first and much, hands next and little, tongue not at all." This is sweeping and sound and applies particularly to the examination of children, but it neglects two important avenues of information—the ears and the nose. It is with the latter organ that I propose to dally a little.

Once an honorary in a hospital ward was remarking on the various clues one might use in making a "spot diagnosis" of a patient's malady and even of his occupation. A student said: "I could tell you what this patient does for a living not by looking at him, but by smelling him." "Nonsense," replied the honorary, "as he has been in a week and has been thoroughly washed several times." However, the elusive odour of resin proved a true guide to the fact that the man was a timber getter at Warburton.

On another occasion a young and enthusiastic honorary walked into an ambulance room and, without caring to approach the patient, a child obviously *in extremis* with air hunger, remarked to the resident who was admitting the patient: "That child is dying of acute diabetes." How so?" asked the resident. "By the smell of acetone in the breath." The resident uncovered the child, remarking that the sweetish smell was due to the sacks in which she was wrapped and disclosed an obviously acute abdominal condition which proved *post mortem* to be an intestinal obstruction. Before death, however, the urine was found to contain 4% of glucose. The writer was amused on asking a group of fifth year students whether they had smelt a child's breath to see the look of astonishment on their faces and later the evident reluctance with which they made the experiment. Even then some were obviously incredulous of the possible value of such a proceeding.

The ways in which our nose can help us consciously or unconsciously in medical practice are almost legion. On entering a patient's house we

can at once assess its state of cleanliness and the likelihood of its presiding deity being fit to nurse a sick patient. The "heavy" breath of a child who is constipated or who is sickening for some illness, is well known to every mother. Fewer people recognize the slightly "dank" quality of the breath of people who are in the early stages of an ordinary catarrhal "cold." In the early stages of most febrile illnesses in children there is associated with a moist tongue slightly furred in the centre, a not unpleasant breath odour suggesting a mild acetonæmia. This is particularly common in pneumococcal infections, but may appear in the very early stage of acute appendicitis and other febrile conditions. As the appendiceal inflammation progresses to suppuration, gangrene and peritonitis, the tongue becomes dry, brown, furred and the breath becomes "abdominal" in type, slightly suggestive of fæces. If the "pneumococcal" breath and tongue are found at this stage of a suspected appendicitis, it is one point in evidence that the right iliac pain is referred from a right basal pleurisy and pneumonia and not from a suppurating appendix.

It is not often possible to describe smells accurately, as they have neither size, shape nor colour and we are forced to fall back on the unpleasant habit of comparing them with the odours of familiar substances of every day experience.

Osler says of the typhoid fever patient that: "A peculiar odour is exhaled from the skin in some cases" and quotes Nathan Smith in describing it as of a "semi-cadaverous musty character." The modern nurse with her practical mind prefers to call it the "typhoid smell."

Most text-book authors speak of the "acid smelling" sweats so common in acute rheumatism. The offensive smell associated with excessive perspiration in otherwise healthy people has a quality peculiarly its own and is often the reason for seeking medical advice. It may be merely an exaggeration of the well-known "sweaty" smell or it may pass on to that frightful odour associated with fœtid sweating of the feet, axillæ or groins, known as "bromidrosis." To be forced to remain even in the same room with a sufferer from this condition is literally torture to anyone with a sensitive nose.

Those who have nursed typhus fever patients say that patients and wards have a distinctive smell which is a help in diagnosis.

Dr. Scholes, speaking of the diagnosis of moderately severe diphtheria, says: "In many instances there is a distinct acetone-like odour of the breath. In mixed cases the odour is heavy and nauseating." The former, I think, corresponds to the "pneumonic" breath mentioned above and the latter is due to the presence of pus-producing and putrefactive organisms. Later, speaking of the very severe diphtheria, he says: "The odour of the breath is heavy and fœtid." Few practitioners have not made a tentative diagnosis of diphtheria from the odour on first entering the sick room.

The appalling odour of chronic atrophic rhinitis has to be smelt only once never to be forgotten.

Carious teeth can be suspected from the breath of a child or adult without looking inside the mouth.

The peculiar "waxy" smell of chronic otorrhœa suggests a diagnosis even in the absence of visible discharge and sometimes friends seek an explanation of the odour before the patient is conscious of anything amiss.

The odour of the breath of those who wear improperly cared for dentures is such that their camouflaged condition can be guessed at a distance of some feet and one wishes that they would keep yards away or soak their teeth overnight in some suitable deodorant.

The breath in acute diabetes with acetonæmia and to a less degree in other forms of acidosis is sometimes so sweet with acetone bodies as to resemble the "cider-like" smell of ethyl acetate. Woe to the clinician who fails to recognize this odour and make a urinary or blood-sugar examination. The urine, on the other hand, in some cases of acidosis has a strong "ammoniacal" smell, due to the excess of ammonia excreted in the attempt to neutralize the acid bodies. This ammoniacal urine is also noticed in infants who are suffering from a disturbance of fat metabolism, with constipated clayey stools, known in Germany as *"Bilanztörung."*

The urine of children suffering from pyelitis or cystitis may smell "foul" and almost "fæcal" so as to arose the suspicion that the stain on the napkin is from the bowel and not from the urethra. The odour of the urine from adults with cystitis, particularly with retention, may be absolutely "putrid" even when freshly passed. Care must be taken not to mistake the "ammoniacal" smell of garments soaked with stale urine for that indicative of excessive ammonia excretion.

We may next consider stools. A knowledge of the characteristic odour of the various types of stool, particularly in infants, is of great help to the pædiatrician. I cannot do better than quote the following paragraph from L. W. Hill in "Practical Infant Feeding":

> Odor.—The stools of babies fed on milk diet rarely are very offensive. Those of the breast-fed baby have an aromatic acid odor, while those of the bottle baby usually have a cheesy, slightly foul odor. If a high sugar and fat is fed, however, the odor is likely to be slightly acid, even in the normal bottle baby. With sugar and fat fermentation the odor is always acid (acetic and butyric acid), with a diarrhœa caused by protein putrefaction the odor is foul. If the baby is being fed a large amount of starch which he is not digesting well and which is fermenting in his intestine, the odour may be very offensive, like that of a pig-pen. The odor of ammonia is often noticed in connexion with high protein stools, but may in a good many cases be due actually to ammonia on the diaper from an alkaline, concentrated urine rather than the stool.

I think I can add to this that the stools of infants with diarrhœa with much mucus—typically in bacillary dysentry—have a faint "mawkish" smell of body slime which is characteristic.

Who does not know the "beery" or "spirituous" aroma of the ripe alcoholic who drifts or "is shot" into the casualty room and earns the terse epithet of "C_2H_6O" which is so easily inscribed by the

resident? If, however, we wish to make sure that this inscription does not also serve as an epitaph to an undiagnosed fractured base of the skull, we should pay attention to the finer differences of alcoholic effluvia. The roisterer full of recent beer is fairly obvious from the smell of that delightful beverage, but the accident patient who has a short time previous to admission and after the accident been given a pick-me-up of brandy or whisky, may lead to a mistaken diagnosis. His breath will usually smell of fresh unoxidized alcohol and one may proceed to determine the presence or absence of organic injury. The "chronic soaked" alcoholic, on the other hand—particularly the spirits-drinker—will smell of the foul metabolized soakage of many days or weeks. His dry tongue, coated teeth and lips and tremulous condition will usually tell their true story.

In midwifery again the sense of smell is invaluable. The smell of normal *liquor amnii* brings back the memory of many a midnight vigil. The odour yclept "sweet" of the normal lochia during the puerperium may give place to the "heavy" smell indicative of retained secundines or mild saprophytic infection and sometimes to a fouler and less profused discharge in the dreaded streptococcal infection of puerperal septicæmia. Experienced nurses describe a "sweetish" smell which pervades a septicæmia ward.

In gynæcology also our nose may make a diagnosis, when called to a patient suffering from a miscarriage when the soaked rags are hidden under the bed and the "sickly" odour of blood warns us of the condition.

An appreciation of the smell of the various poisonous drugs, such as hydrocyanic acid ("oil of bitter almonds"), camphor, carbolic acid, "Lysol" and the various strong acids, chloroform, ether and so forth, may be of use in emergency and may lead to prompt, correct treatment when inquiry might be fruitless or too late.

To turn from medical to surgical diseases we have the horror of the all-pervading odour of moist gangrene, which can be almost unbearable in an enclosed space, may fill the house and on a still day spread up and down the street. To what extent an experienced surgeon can diagnose the causative organism from the smell of pus is largely a matter for his conscience, but every student knows the "ah" with which the surgeon sniffs at the first exploring swab which comes to light with the stain and characteristic "fæcal" smell of *Bacillus coli communis* pus. Absence of smell or a faint "sweetish" smell of pus evacuated from a bone or gland abscess is a point in favour of its being tuberculous. The staphylococcal pus of boils and skin abscesses has a faint "nauseating" smell. Another surgical condition in which smell plays an important part is in the diagnosis of obstructive vomiting, at first gastric, then bilious and later sometimes fæcal. The obviously "fæcal" smell can be recognized before the nature of the vomited material can be certainly diagnosed by any other means.

In bronchiectasis, abscess and gangrene of the lung the *crescendo* of foul odours tells its own story.

Finally, experienced nurses and physicians recognize a very definite exhalation with approaching dissolution for some time—even some hours—before death and know of a surety that the sufferer is approaching "the first dark hours of nothingness—the last of danger and distress."

THE ESTIMATION OF THE SALIVARY UREA AS AN INDEX TO RENAL PROGNOSIS.

By A. B. Corkill, M.B., B.S.,
From the Bio-Chemical Laboratory, Alfred Hospital, Melbourne.

Tests of renal functions fall into two main categories: (i.) Those associated with the detection of excess metabolites in the blood, for example urea, uric acid, creatinine; (ii.) those associated with a diminished excretory power toward certain drugs, for example, phenolphthalein, urea.

MacLean has shown[1] that the urea concentration test together with the blood urea test, gives practically all the information that is required. He has drawn attention to the fact that it is possible to influence the blood urea by a low protein diet.[2] He cites the instance of a patient with a blood urea of one hundred and twenty milligrammes, which was reduced to thirty on a low protein diet, mainly carbohydrate. However, provided the above fact is recognized and the blood urea is estimated in conjunction with an ordinary diet, it should prove a consistent and valuable index to renal function.

The present methods in vogue for the determination of blood urea are chiefly those dependent on the urease ferment of the Soy bean. This ferment has the power of converting urea into ammonium carbonate. Addition of an alkali to the latter liberates ammonia which is aerated through a known amount of one hundredth normal sulphuric acid and from this the amount of urea can be calculated.

Although quite a simple laboratory procedure, the above estimation is not absolutely practicable for the general practitioner. It is not worth his while to set up apparatus and solutions for a test which he will require only occasionally to perform. Nevertheless, there are occasions when it is essential to have some idea of the blood urea concentration. The method to be described has the following advantages:

(1) It is simply and quickly carried out;

(2) No elaborate solutions are required and those used will keep indefinitely;

(3) As no blood is required, repetition of the test may be frequently carried out without any inconvenience to the patient.

Principles Underlying the Estimation of the Salivary Urea.

Hench and Aldrich have shown[3] that urea in the body is extremely diffusible and has a definite concentration in the body fluids. They have further demonstrated that it is present in saliva and can be easily estimated.

In addition to urea saliva contains ammonia and Hench was able to show that its presence was due

to oral bacterial action upon the urea and, therefore, should be considered as part of the urea. It follows that ammonia nitrogen and urea nitrogen should be considered as strictly comparable to the blood urea nitrogen. In actual practice it is found that the combined salivary urea nitrogen closely follows the blood urea. In cases of blood urea retention there is a simultaneous rise in the salivary urea.

Mercuric chloride in excess together with saturated sodium carbonate solution gives a reddish-brown precipitate of mercuric oxy-chloride. Certain bodies in the saliva have the power of combining with mercuric chloride; of these the greatest affinity is possessed by the urea. This fact may be expressed in an empirical way by stating the combining power of one hundred cubic centimetres of saliva in terms of 5% mercuric chloride. This factor Hench has termed the salivary urea index and for normal persons it lies between thirty and fifty cubic centimetres of 5% mercuric chloride for one hundred cubic centimetres of saliva.

Apparatus Required.

The following apparatus is required: A small flask of about one hundred cubic centimetres capacity; a white porcelain plate with small depressions, each to contain a drop of the saturated sodium carbonate solution; a twenty-five cubic centimetre burette; a saturated solution of sodium crabonate; a 5% solution of mercuric chloride; a small glass rod to test a drop of the saliva against the sodium carbonate.

Collection of the Saliva.

About eight to ten cubic centimetres of saliva are required. The mouth is first washed out with water to remove food particles. To promote the salivary flow paraffin wax can be chewed. Chewing gum should not be used. Two specimens of about eight cubic centimetres are collected and the first discarded. From the second specimen five cubic centimetres are pipetted off into the flask.

The Titration.

The burette is filled with 5% solution of mercuric chloride and on each of the depressions of the porcelain plate is placed a drop of the saturated sodium carbonate solution. From the burette the mercuric chloride is run into the saliva. At first one cubic centimetre may be safely run in and then the addition is proceeded with five drops at a time. The addition of mercuric chloride is continued until one drop of the mixture added to a drop of sodium carbonate of the porcelain plate causes an immediate reddish-brown precipitate. This end point indicates that all the urea in the saliva has been used and the excess mercuric chloride is now combining with the sodium carbonate to form the reddish-brown oxy-chloride. The result may be expressed as the number of cubic centimetres of mercuric chloride solution required for one hundred cubic centimetres of saliva and is termed the salivary urea index. Normally it lies between thirty and fifty. A somewhat approximate idea of the blood urea may be obtained from the following formula:

$$x = (S \times 1.43) - 34.$$

x is the probable blood urea, S = the salivary urea index. If the salivary urea index were 100

$$x = (100 \times 1.43) - 34 = 109.$$

Record of Cases.

Firstly an attempt was made to determine the normal variations of the salivary urea index. The figures arrived at closely approximated those given by Hench for one hundred cubic centimetres of saliva between thirty and fifty cubic centimetres in terms of 5% solution of mercuric chloride. The following figures picked out at random will illustrate this:

Number of Patient.	Salivary Index.	State of Patient.
1	40	Healthy student
2	30	Healthy student
3	44	Healthy student
4	35	Healthy student
5	42	Healthy student
6	50	Rheumatoid arthritis
7	20	Rheumatoid arthritis
8	37	Gastric ulcer
9	30	Diabetes insipidus
10	44	Convalescent empyema

Apparently the upper limit lies about fifty corresponding approximately to the upper limit of the blood urea. The normal variations having been ascertained, investigations were then carried out in a number of cases of suspected renal insufficiency. It must be remembered that the formula given to obtain the probable blood urea from the salivary urea is only an approximate one. However, it may be pointed out that our figures always were within four to eight of the actual blood urea and hence for all practical purposes it serves as a valuable indication of the renal condition. Fluctuations in the blood urea will be shown by simultaneous variations in the salivary urea index.

CASE 1.—M.C., *ætatis* fifty-eight years, suffered from an enlarged prostate of some years' duration.

Blood urea	72 milligrammes
Salivary index	79
Probable blood urea	78 milligrammes

Supra-pubic drainage was performed and a subsequent estimation revealed the following:

Blood urea	46 milligrammes
Salivary index	58
Probable blood urea	50 milligrammes

CASE 2.—D.C., *ætatis* seventy years, was admitted on October 30, 1924, in a comatose condition. No localizing signs of any description were present. The diagnosis was either uræmia or cerebral thrombosis.

There was no euglobulin present in the cerebro-spinal fluid, while the total albumin content was 0.03%.

Blood urea	38 milligrammes
Blood creatinine	1 milligramme per 100 cubic centimetres
Salivary index	50
Probable blood urea	37.5 milligrammes

CASE 3.—Three years ago M.M., *ætatis* twenty-years, had acute nephritis. He was well until three weeks ago; he then complained of failing vision, first in the right eye and later in the left. For seven days he has had severe frontal headaches, bilious feeling and dancing lights in front of the eyes.

His blood pressure is 235 millimetres of mercury. His arteries are somewhat thickened. There are definite papillœdema and retinal hæmorrhages.

The urea concentration test yielded 1.03% during the first hour and 1.21% during the second hour.

Blood creatinine	2.6 milligrammes
Blood urea	121 milligrammes
Salivary index	110
Probable blood urea	123 milligrammes

CASE 4.—V.T., *ætatis* fourteen years, was admitted on November 4, 1924, complaining of weakness and general malaise. Two weeks ago she vomited and since then she has had headaches and a feeling of nausea.

All signs of acute nephritis were present. On November 10, 1921, the result of the urea concentration test was in the first hour 1.5% and in the second hour 1.45%.

Blood urea, 90 milligrammes.

On November 19, 1924, the result of the urea concentration test was in the first hour 1.2% and in the second hour 1.35%.

Blood urea	64 milligrammes
Salivary index	72
Probable blood urea	69 milligrammes

Conclusions.

1. The determination of the salivary index is a simple test and quite within the scope of anyone not possessing laboratory facilities.

2. From it a working idea of the actual blood urea may be deduced.

3. In terms of 5% mercuric chloride the salivary index normally lies between thirty and fifty cubic centimetres per hundred cubic centimetres of saliva.

References.

(1) Hugh MacLean: "Modern Methods in Diagnosis of Renal Disease."
(2) Hugh MacLean: Ibidem, Chapter IV.
(3) P. S. Hench and M. Aldrich: "The Concentration of Urea in Saliva," *The Journal of the American Medical Association*, October 21, 1922, page 1409; *Medical Clinics of North America*, Volume VII., 1923, page 123.

Reports of Cases.

MELÆNA IN A BABY THREE DAYS OLD.

By ELLEN M. KENT HUGHES, M.B., B.S., (Melbourne),
AND
N. E. DAVIS, M.B., Ch.M., (Sydney),
Kingaroy, Queensland.

A *multipara* gave birth to her tenth child, a healthy female, on September 5, 1924. On the morning of the third day the nurse reported that the napkin was stained with blood. Parke Davis and Company's "Hæmoplastin" was administered every four hours in doses of 0.5 cubic centimetre, but the child continued to hæmorrhage. In the evening she appeared exsanguinated, was almost pulseless and obviously dying. Thirty cubic centimetres of blood were withdrawn from the father and injected into the buttock of the child. Hæmorrhage occurred during the operation, but bleeding ceased five minutes afterwards. She slept peacefully and except for passing eight cubic centimetres of old blood at 7 a. m. next morning. There was no further trouble.

In regard to the hypothesis advanced by some that hæmorrhage in the newly born is due to an infective condition, it is interesting to note that the mother had a severe attack of mumps during the eighth month of pregnancy and tonsilitis during the ninth month. A week after her confinement she suffered from a breast abscess. All her previous pregnancies and confinements had been normal. At the age of two and a half months the child was still pale, but was making good progress.

MELÆNA NEONATORUM IN A MALE INFANT THIRTY-SIX HOURS OLD.

By A. J. MAURICE PURCHAS, M.B., Ch.M. (Sydney),
Young, New South Wales.

ON January 25, 1925, an apparently healthy *primipara* was delivered of an apparently healthy male infant, the presentation was persistent occipito-posterior, F. M. Purchas's modification of Milne-Murray's forceps was used. There were marks of the forceps below both ears.

No history of infective condition was to be obtained. There were no evidences or reasonable suspicion of *lues* in either mother or father.

The child appeared normal until its thirty-sixth hour of life when the nurse reported to me that it had vomited a large amount of dark blood and had passed a profuse motion of pure blood. At the end of four and a half hours from the onset, the child had passed five large motions of blood and had vomited blood three times. Its condition was precarious, the skin being blanched, cold and clammy, the pulse being soft and uncountable, the respiration being irregular and sighing. During this interval adrenalin solution given by mouth and by rectal injection and two thousand units of concentrated antidiphtheretic serum given subcutaneously both failed to stop the hæmorrhage.

As the condition of the baby allowed no time for even the roughest typing of blood, I took upon myself the risk of incompatibility and injected three cubic centimetres of whole mother's blood, taken from the median basilic vein, subcutaneously into the child's flank.

The bleeding ceased immediately, only one small motion of stale blood being passed several hours later.

During the subsequent four weeks the baby showed no ill-effects from either injection and, though still pale and icteric, made slow progress. A huge abscess appeared below the angle of the left mandible necessitating incision.

At the time of treatment the method was, as far as my knowledge extended, original, but I find that Porter and Carter on pages 118 and 498 of their book, "Management of the Sick Infant," mention intra-muscular injection of whole blood as the best treatment. They advise the use of father's blood and state that the blood need not be grouped, but do not recommend the injection of any definite quantity.

NOTES ON A CASE OF DISSEMINATED FAT EMBOLISM.

By ARTHUR A. PALMER, M.B., Ch.M., F.R.C.S. (Edin.),
Government Medical Officer, Sydney;
AND
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Principal Microbiologist, Department of Public Health, Sydney.

THE following particulars of a case of fat embolism may be of interest in the light of the preceding paper by Dr. Corlette.

For the clinical history we are indebted to Dr. H. K. Porter, at the time Medical Superintendent of Sydney Hospital.

The *post mortem* examination was carried out by one of us (A.A.P.) at the city morgue and the histological examinations were made at the Microbiological Laboratory, Department of Public Health.

Clinical History.

J. H., male, aged thirty-nine years, wharf labourer, was admitted to Sydney Hospital on March 13, 1923, after having been crushed by a stack of chaff falling on him.

On examination a compound fracture of the right tibia was found, about midway between the ankle and the knee, with the lower fragment projecting several centimetres through the skin. The fibula was broken just above the tip. The cardiac, respiratory and nervous systems were clear. He was operated on by Dr. R. Bridge the same day under ether anæsthesia. The wound was cleaned, the ends of bone were approximated and

kept in apposition with a plate, the wound was loosely sutured and 1,500 units of anti-tetanic serum were given. Twenty-four hours later the patient appeared drowsy and listless. The knee jerks were absent; the left plantar reflex was definitely of the Babinski type. Neither Kernig's sign nor head retraction was present and the wound was clean.

On March 15, 1923, the urine contained a heavy cloud of albumin. The patient was comatose.

On March 20, 1923, the patient died in coma.

In addition to the condition described in the above notes, we were informed at the time of the autopsy that the patient had had several fits before he lapsed into coma.

Post Mortem Notes.

Autopsy was performed on March 20, 1923. The tibia was found plated, the fragments were in good condition. Numerous petechiæ were present on the skin. Numerous similar small hæmorrhages were seen beneath the capsules of the kidneys, while hundreds of similar areas were scattered throughout the white matter of the brain.

The upper lobes of the lungs were bright and mottled; the lower lobes were dark, the appearance resembling that seen in death from pneumonic influenza, except that there did not appear to be any consolidation.

A note made by one of us (E.W.F.) at the time of the *post mortem* examination was to the effect that the lungs were partially collapsed and of a deep plum colour at the base. The liver was noted as being pale with (?) hæmorrhages.

Histological Appearances.

Sections of the brain, lung, kidney, liver and skin were cut by the freezing method. These sections were stained with Sudan III.. Further portions of the same tissues were fixed in the usual manner, embedded in paraffin and the sections stained by Heidenhein's iron-hæmatoxylin method.

Brain.

Examination of frozen sections of the brain stained by Sudan III. revealed the presence of fat droplets in the capillaries, especially near the surface. The hæmatoxylin sections contained evidence of congestion with small hæmorrhages.

Kidney.

In the Sudan III. sections of the kidney fat droplets, particularly in the glomeruli, were seen.

In the hæmatoxylin sections small hæmorrhages into the convoluted tubules and Bowman's capsule, particularly near the surface of the cortex, were found.

Lungs.

In frozen sections the alveoli appeared partially collapsed and contained a cellular exudate; fat droplets were present in the capillaries of the alveolar walls.

The paraffin sections manifested similar appearances in the alveoli, but hæmorrhages were noted into the lumina and the capillaries were the seat of intense engorgement.

Liver.

Advanced fatty infiltration of the liver was seen in sections stained by both methods.

Skin.

In the frozen section of the skin fat droplets were present, but it was sometimes difficult to distinguish between fat normally present in the subcutaneous tissues and true fat emboli. The presence of fat droplets in the capillaries was, however, held to be evidence of fat embolism. Hæmorrhages were present in the subcutaneous tissue and in the corium.

CEREBRAL FAT EMBOLISM.

By STRATFORD SHELDON, M.B., Ch.M., (Sydney),
Sydney.

I HAVE read the proof of Dr. Corlette's paper on cerebral fat embolism and his reference to a recent case of which I made a *post mortem* examination for the Coroner. I did not recognize that this was a case of cerebral fat embolism, but thought it was a hæmolytic condition, the result of some infection.

Mrs. M. aged twenty-two years, was injured in a ferry accident on August 28, 1924. She died on August 31, 1924, after being unconscious for many hours before death. Her injuries were not particularly severe and as there was no injury to the head, the reason of her being unconscious was obscure.

The injuries consisted of a bruise on the inner side of her right elbow and slight bruising of her left foot. A rib was broken on the left side and the junction of the manubrium and the sternum was broken with bruising in the mediastinum behind it.

There was a peculiar thick petechial rash all over the upper part of the trunk, a similar condition on the surface of the lungs and on the visceral pericardium. There was no consolidation of the lungs. The uterus was subinvoluted. She had been confined three weeks previous to the accident and there was some grey, brown decidua in the fundus of the uterus.

No microscopical examination was made and as I have said before, I did not suspect the possibility of fat embolism.

Reviews.

TREATMENT BY "INSULIN."

THE perusal of the second edition of "The Insulin Treatment of Diabetes Mellitus" by Dr. P. J. Cammidge should supply a useful stimulus to those who are tending to drop into a routine method of using this new remedy.[1] Condensed into a small volume is here found such a mass of information about "Insulin" and its action in various disturbances of carbo-hydrate metabolism, that intending readers must be prepared for some very solid thinking before they can hope to assimilate it. The elaborate nature of the methods of investigation advised by the author is such as can only be followed by those who have access to an extremely well equipped bio-chemical laboratory. In actual practice in Australia the cost of the investigation alone would, we fear, be prohibitive in the case of most of the patients met with in private practice. But even if all the tests recommended cannot be carried out in every case, it is an education in itself to know what others are doing and how help may be obtained in at least the more difficult cases.

Much of the material that has been added to the first edition has been collected into an *addendum* and special interest attaches to the chapter on "Insulin" in the treatment of non-diabetic conditions.

Surgeons who have experienced the anxiety incident to operations upon emaciated persons unable to be fed by the ordinary channels, will read with interest the paragraphs dealing with the improvement obtained in such people after intravenous injections of glucose solutions simultaneously with subcutaneous injections of suitable quantities of "Insulin."

ULTRA-VIOLET RADIATION.

WE have received a work by Dr. Percy Hall on ultra-violet rays in the treatment and cure of disease.[2] This work should stimulate the interest of medical practitioners in this sadly neglected branch of the profession. In the first three chapters the author deals with heliotherapy generally and refers to the curative effect of sun-

[1] "The Insulin Treatment of Diabetes Mellitus," by P. J. Cammidge, M.D. (London), D.P.H. (Camb.); 1924. Edinburgh: E. & S. Livingstone; Crown 8vo., pp. viij. + 172. Price: 6s. net.

[2] "Ultra-Violet Rays in the Treatment and Cure of Disease," by Percy Hall, M.R.C.S. (England), L.R.C.P. (London), with Introductions by Sir Henry Gauvain, M.A., M.D., M.C. (Cambridge), and Leonard E. Hill, M.B. (London), F.R.S.; 1924. London: William Heinemann (Medical Books), Limited. Royal 8vo., pp. 110, with illustrations. Price: 7s. 6d.

light in many conditions and refers especially to the wonderful results obtained in the treatment of tuberculosis of joints and bones and of infantile rickets. Therapeutically, radiant energy is divided into (i.) radiant heat produced by artificial means, (ii.) helio-therapy or the action of the heat and light rays of the sun and of the electric arc, (iii.) ultra-violet radiation, (iv.) the action of X-rays and radium. Experiments in Vienna and elsewhere have shown that children and rats which are fed upon a low vitamin diet, will, if exposed to ultra-violet radiation, thrive just as well as those fed on full vitamin diet. These experiments show that by this means deficiency of vitamin diet can be counteracted by ultra-violet radiation. Ultra-violet rays are bactericidal and discharging wounds and ulcers exposed to the sun's rays in the Alps, where ultra-violet rays are present in large quantity, heal with rapidity.

In the next four chapters the production of ultra-violet rays from arc and mercury vapour lamps is described; the author points out that glass and ordinary water arrest the passage of ultra-violet rays, while quartz is practically non-absorbent. He also describes the various arc lamps obtainable for this work, including the lamps of Reyn, Finsen and others, but the most suitable lamp available is the tungsten arc lamp which is described in detail. The tungsten arc light is richest in ultra-violet rays and the amount of current necessary is only from five to ten ampères as against the eighty ampère current needed for the Finsen and other lamps. Mercury vapour lamps have not been a success in the author's experience. In the eighth chapter he describes the best form of tungsten arc. Direct current is necessary and suitable controls for varying the ampèrage and voltage. Reflectors are useful and should be nickel plated. The arc should not be filtered, but a cold air stream may be directed upon the part under treatment in order to prevent burning by the heat rays.

Operators should cover the face and wear blue glasses; the rest of the body is sufficiently protected by the clothing.

Blondes react quickly and the younger the patient, the quicker is the reaction. Olive oil may be applied to relieve irritation, but this is not great unless the patient washes soon after treatment. Erythema is the guide to treatment. Too violent a reaction is to be avoided.

Exposures usually last for two minutes at fifty centimetres distance with five to seven ampères in circuit; the dosage is repeated when reaction has ceased. Chronic skin lesions react rapidly to ultra-violet radiation. Rheumatic disorders, myalgia, fibrositis, arthritis and lumbago are sometimes completely relieved after a couple of doses. The author has seen some remarkable cures in cases of neurasthenia, large areas of the trunk being exposed at one sitting.

Benefit is obtained in catarrhal conditions of the air passages from ultra-violet radiations. Infantile rickets and surgical tuberculosis are very suitable affections for treatment by this method. Case reports are included at the end of the various chapters. The results obtained certainly warrant more extensive use of these ultra-violet radiations.

SURFACE ANATOMY.

THE latest edition, the sixth, of Dr. L. Bathe Rawling's "Landmarks and Surface Markings of the Human Body" follows closely on the fifth edition, of which there were seven reprints.[1] This speaks volumes for this handy little manual, very useful alike to student and practitioner.

In Chapter I., dealing with cranio-cerebral topography, the author opens by stating: "Only those landmarks and surface markings will be given which are of practical value and, as far as possible, each landmark will be rendered independent of any other, as by such means any given structure can be rapidly depicted on the surface, the important question of time and of space rendering the more complicated systems in which it is necessary to map out a network of intersecting lines in order to fix the position of any single structure, of little surgical value.'

This is well carried out and is a point of some note as regards the topography of this region. The edition is excellently illustrated by over thirty diagrams and is concise, the whole volume comprising only ninety-three pages of valuable reading. There is no "padding," every line conveying important information on the point of issue. The volume is well set up, information on any particular point being easily ascertained without loss of time.

In the appendix is given "the length of passages, tubes" *et cetera*, "the weight of some organs" and "the ossification and epiphyses of the bones of the upper and lower extremities."

Altogether the book contains ninety-three pages of condensed and useful information and is well worth its place in any medical library.

[1] "Landmarks and Surface Markings of the Human Body," by L. Bathe Rawling, M.B., B.C. (Cantab.), F.R.C.S.; Sixth Edition; 1924. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. viii. + 98, with 36 illustrations. Price: 7s. 6d. net.

DIFFERENTIAL DIAGNOSIS.

THE third edition of "Differential Diagnosis" by Dr. Cabot (Volume II.) is an amplification of the original edition which was published in 1914; a great deal has been revised and modified in order to bring the publication up to date.[1]

It will be remembered that the first volume of this book was issued in 1911; the author dealt in it with pain and other symptoms, twelve main symptoms in all, and at the time it was very favourably commented on by reason of the novel method used of imparting clinical instruction.

The fact that the teaching is clinical and the methods used are not infallible appealed at once to students of medicine. The author states in his preface to the first volume that "the attempt to make and defend a differential diagnosis brings all one's failings into sharp relief," a statement with which every seasoned practitioner will agree.

In the second volume he deals with nineteen different subjects, or perhaps it should be said symptoms and to each of these a chapter is devoted.

The symptoms are those with which the practitioner is familiar: abdominal and other tumours, vertigo, dyspepsia, diarrhœa, hoarseness, pallor, fainting, every day subjects presented to make a fascinating and complete study.

The plan of the book is such that at a glance the relative frequency of the disease causing the symptom can be seen from the table record of each symptom; this is followed by a *résumé* of the conditions giving rise to the symptoms coming under discussion.

Some of the statistics are surprising. For example, it would hardly be thought that there were nearly six times as many causes of dyspepsia which are not gastric as causes which arise in the stomach itelf. Yet, taking this tabular record of dyspepsia alone and leaving out those causes which cannot be classified, it is two and a half as common as a result of failing heart than as a result of gastric ulcer and gastric ulcer is the commonest cause of dyspepsia in a truly gastric sense mentioned in the table.

A wealth of very necessary and carefully compiled detail is given and the various steps taken to arrive at a diagnosis are described, together with a frank discussion on the points which arise in each type of disease.

The history of each patient is then followed until the diagnosis is confirmed on the operating table or in the *post mortem* room and the facts are plainly and vividly recorded.

The book is to be commended. To those who are unable to visit large hospitals or schools for the purpose of post-graduate study, it bridges or helps to bridge a wide gap. While it cannot be said in any sense to take the place of clinical study, it is a worthy effort to bring modern clinical ideas to every practitioner.

[1] "Differential Diagnosis," by Richard C. Cabot, M.D.; Volume II., Third Edition, Revised; 1924. Philadelphia and London: W. B. Saunders Company; Melbourne: James Little. Royal 8vo., pp. 709, with 254 illustrations. Price: 45s. net.

The Medical Journal of Australia

SATURDAY, MARCH 7, 1925.

A Popular Medical Magazine.

THE daily newspapers seem to delight in publishing paragraphs and articles dealing with medical matters. In Australia these paragraphs and articles are not submitted to competent medical practitioners for revision, with the result that save on quite exceptional occasions the news conveyed is misleading and often utterly absurd. For many years *The Times* has employed a medical man of eminence who attends at Printing House Square every day to handle all medical and hygienic matters. It has been worth the proprietors' while to pay this medical correspondent a handsome retaining fee for this work and the result has been excellent. In Australia newspapers have not recognized the advisability of paying for accuracy in technical matters of this kind. The public in consequence obtains an entirely false record of modern achievements in medical science and not infrequently suffers real damage as a result of sensational statements devoid of a reliable basis. Science can be entrancingly interesting and fascinating, but it cannot be sensational. It is necessary to counter this cheap form of journalism.

The daily newspapers also contain from time to time articles on medical subjects written and signed by medical practitioners. The General Medical Council has repeatedly warned doctors in active practice against this. In recent times special attention has been directed toward this form of "oblique" advertising and a special notice has been issued, warning offenders that the General Medical Council in Great Britain will remove their names from the medical register. It is eminently desirable that the public should have reliable information on certain medical matters, but the greatest care should be exercised concerning the manner in which this information is conveyed. There are many pitfalls to be avoided and not the least of these is the boosting of hypotheses sponsored by one individual and not or not yet accepted by a substantial section of the medical profession.

The proposal has been made by the New South Wales Branch of the British Medical Association that steps be taken to establish a popular magazine which could be employed for the purpose of instructing the people generally on the nature of disease processes, on the problems connected with the preservation of health and the prevention of disease, on the progress of pathological and biochemical science and on similar matters. The idea has commended itself as practicable and advantageous to the several Branches of the British Medical Association in Australia and the Federal Committee has invited the Australasian Medical Publishing Company, Limited, to undertake the establishment of such a magazine. The Directors of this company are prepared to accept this invitation. It is probable that a monthly medical magazine will be issued in a short time.

In order that an undertaking of this kind may achieve immediate success it is essential that three things should be carried into effect. In the first place the magazine itself must be constructed on sound and sensible lines. The articles must be attractively written, must be authoritative and must be in accordance with the best modern teaching. The language employed must be free from unnecessary technicalities and elaborate details must be avoided. Good illustrations should be utilized, for everyone loves a picture book. In this connexion the choice of articles and of writers should be in the hands of a competent editorial committee and all contributions should be anonymous.

The second essential to success is that the medical profession should recommend this magazine to lay persons of all classes and callings. It would be freely advertised, so that the public would become familiar with its name and external appearance and the medical profession would be required to guarantee its authority.

In the third place the Councils of the six Branches of the British Medical Association in Australia should use its columns to inform the public

what the Association is, what it does and how it does it. The calumnies and mispresentations so often printed and spoken of this great organization should be frankly and fearlessly met and contradicted. Many people would thus be induced to buy the magazine in order to learn what the Association has to say in self-defence. If the remaining contents were attractive, interesting and helpful, the popularity of the publication would soon be assured. We venture to direct the attention of the medical profession to this subject at this stage, so that useful propaganda work may be carried out in advance.

Current Comment.

METASTATIC TUMOURS OF THE THYREOID GLAND.

A MALIGNANT tumour may be defined as a collection of cells undergoing limitless, disorderly growth, endowed with power to invade surrounding tissue and infiltrate it. These cells when carried as emboli in the blood or lymphatic stream, form metastatic deposits which in their turn grow and infiltrate the tissues of their new resting place, but nevertheless retain their original morphological characteristics. Metastases may result from tumours of all grades of malignancy and it may be exceedingly difficult to determine whether or not a tumour is malignant. At the same time it is extremely unlikely that metastases can take origin from a structure of a benign or non-malignant nature. This has been held to occur, however, in the case of some secondary tumours of bone whose cells present the appearance of normal thyreoid tissue. The question of thyreoid metastases in bone has been discussed recently by Mr. F. Gordon Bell.[1]

Mr. Bell refers particularly to secondary thyreoid tumours of bone which are found when the thyreoid gland presents or appears to present no departure from the normal detectable on physical examination or when it is the seat of a colloid or adenomatous change. He refers to the influence of Cohnheim in the interpretation of these as examples of benign metastatic goître and states that if this interpretation be accepted, it allots to the thyreoid a unique place in the range of surgical pathology. He weighs the evidence which may be taken to support the Cohnheim hypothesis. In the first place the thyreoid possesses a blood supply which is capable of great variation, the circulation between the alveoli is of a sinusoidal character and the glandular epithelium is brought into an unusually intimate relation with the vascular endothelium owing to the absence of a basement membrane. The thyreoid cell possesses exceptional potentialities in regard to size, number and functional activity. It may thus be rendered more mobile and in the absence of a basement membrane pierce the slender barrier that separates it from the blood stream. Having entered the blood stream, the cell may possibly be carried as an embolus to another part of the body, become grafted on a new tissue and undergo proliferation. Errors in development and the osseous inclusion of aberrant thyreoid tissue offer a plausible explanation for some benign tumours in association with which no obvious changes in the thyreoid gland can be found. Mr. Bell regards such an explanation as possible with tumours in the neck and adjacent areas. He does not think, however, that a developmental origin can be alleged in the case of the long bones, pelvis, ribs, skull and lower part of the spine. In these situations tumours must be assumed to have arisen from detached thyreoid cells carried by the circulation. The chief evidence in favour of metastasis from a normal or benign thyreoid is derived from reports of cases in which a thyreoid tumour in bone has been associated either with a clinically normal thyreoid or with some benign condition, such as colloid or adenomatous goître. The first of these two groups is small and according to Mr. Bell must be accepted with great reserve in view of the fact that the condition of the thyreoid gland has not been determined beyond doubt by an adequate microscopical examination. There is no doubt that Mr. Bell is right in his contentions. Most medical practitioners can recall cases in which malignant disease of the breast, for example, has been found only after complete serial sections of the organ have been made. The second group in which a metastasis from a colloid or adenomatous goître is postulated, is a much larger one. Mr. Bell states that many of these cases are open to criticism. He refers to Cohnheim's classical case. In this instance it was found that portion of a colloid growth had penetrated the wall of one of the inferior thyreoid veins. Under such circumstances transplantation of thyreoid tissue would readily occur. But Mr. Bell states that it would be necessary to assume an abnormal growth activity on the part of the colloid goître to enable it to penetrate the wall of the vein. He quotes the opinion of Wölfler that "when the glandular cells distributed by metastasis have given rise, in different situations, to tumours which not only grow but exert a destructive action on the bone, the primary tumour should be considered benign neither clinically nor anatomically, even if its malignancy cannot be established by microscopic examination."

Mr. Bell gives details of three cases investigated by himself. The first patient had a malignant goître and also a secondary deposit in a rib. The second patient suffered from simple enlargement of the thyreoid gland for a number of years. One lobe increased rapidly in size and was found to have undergone early malignant change. A secondary deposit occurred in the sternum two years later. The third patient presented no abnormality of the thyreoid gland during life. Her femur (she was a woman, aged forty-eight years) fractured spontaneously while she was lying in bed. Great

[1] *The British Journal of Surgery*, October, 1924.

enlargement of the left ilium was present. The thyreoid nature of the lesions was not recognized until microscopical examination was made at autopsy. It is unnecessary to refer to these cases in more detail. Mr. Bell in discussing them shows that superficial inspection of the thyreoid at autopsy may easily fail to reveal minute changes within the gland and he points out further that a secondary thyreoid tumour in bone of unquestionable malignant nature may display a structure, fœtal or adult, hardly if at all distinguishable from that of a normal or benign gland.

The question raised by Mr. Bell is one of extreme interest to morbid anatomists. Although it may be possible theoretically for cells of a benign growth to enter the blood stream and to become grafted on to tissues in another part of the body, it would be necessary to undertake most complete microscopical examinations in a long series of cases before it could be recognized that such an unusual condition occurs. The examination would necessarily include serial sections of the whole thyreoid gland in question and the discovery of areas in regard to which doubt was entertained would be sufficient to exclude the specimen from those regarded as benign.

MELIOIDOSIS.

In July, 1922, attention was drawn in this journal to investigations into a new disease known as tularæmia which had been described in America. Tularæmia occurs among rodents and it is supposed that the infection is conveyed to man by the bites of insects. We pointed out that though no case of tularæmia had been reported in Australia, conditions favourable to its occurrence existed in the Commonwealth and that for this reason a knowledge of its characteristics was important. For the same reason it is desirable that medical practitioners, particularly those in the tropical parts of Australia, should be acquainted with the main features of another disease of rodents which sometimes affects man.

In 1912 Whitmore and Krishnaswamy reported the occurrence of a new disease which presented many similar features to glanders and in 1913 Whitmore isolated and described a bacterium in connexion with it. Dr. A. T. Stanton and Dr. William Fletcher have recently given a very clear account of this condition.[1] They described it in 1921 and chose for it the name melioidosis because the physicians of ancient Greece described under the name μέλι a variety of conditions resembling glanders. The causative organism has been found in the blood, sputum, urine and intestinal contents of man and animals. Animals have been infected by subcutaneous puncture or by introduction into the nares by a platinum loop or into the gastro-intestinal canal in the food. The manner in which man is infected, has not been determined. A large proportion of individuals affected were morphine injectors, the result was that the disease was sometimes known as "morphine injectors' septicæmia." Drs. Stanton and Fletcher, however, regard it as most likely that man is infected by the ingestion of food which has been contaminated by rats, on account of the severity of gastro-intestinal symptoms. The causative organism, *Bacillus whitmori*, as seen in smears from lesions in an infected organ, is about the size and shape of *Bacillus mallei*. It stains readily with the usual stains, but is neither acid-fast nor Gram-positive. With Leishman's stain it presents bipolar staining. Growth upon all the usual culture media is rapid and luxuriant; it occurs under both aerobic and anaerobic conditions, though much more luxuriantly under the former. Two prominent features of *Bacillus whitmori* are its motility in young cultures and the fact that when grown on glycerine agar it assumes a heaped-up and rugose appearance. The organism also liquefies gelatine and curdles milk.

The characteristic lesion of melioidosis is a small, yellow, caseous nodule which commences as a collection of polynuclear cells surrounded by congestion. By the time the nodules have become large enough to be visible to the naked eye they consist of a mass of necrotic caseous material containing a great number of chromatin fragments which are the remains of dead nuclei. The nodules have been found in every part of the body except the brain and they coalesce to form large areas of caseation, particularly in the lungs. These areas sometimes break down in the viscera of man to form abscesses.

The symptoms produced in melioidosis depend on the virulence of the infecting strain, on its initial dose, on the resistance of the patient and on the part of the body affected. The clinical picture may be that of a septicæmia or it may resemble that of cholera or plague. In less virulent forms the patient may lapse into the typhoid state or his condition may resemble that of generalized tuberculosis. If pustules or abscesses predominate, a diagnosis of acute glanders may be made. If the patient lives long enough, bone lesions may make their appearance and these may resemble condition associated with tertiary syphilis or chronic tuberculosis. Delirium is commonly present and Drs. Stanton and Fletcher report the case of a patient who was confined in a cell because he was supposed to be suffering from acute mania.

The prognosis is exceedingly grave. With two exceptions, every person known to have been affected with melioidosis, has died. One of those who recovered, became a chronic invalid owing to the presence of abscesses, sinuses and chronic suppuration in the bones of the feet. The disease does not appear to be communicable to healthy individuals by patients. No segregation has been practised and no instance of transmission of the disease from one person to another has been recorded. So far fifty cases only have been noted as occurring in human beings, but Drs. Stanton and Fletcher lay stress on the fact that all great epidemic diseases must have had small beginnings and that it is necessary to be on guard against such a painful and fatal malady.

[1] *The Lancet*, January 3, 1925.

Abstracts from Current Medical Literature.

MEDICINE.

Migraine.

A. F. HURST (*The Lancet*, July 5, 1924) gives his experiences of the ætiology and treatment of migraine. The syndrome has been recognized since the second century and various hypotheses have been advanced as to its cause; anaphylaxis, disorders of internal secretions, excess of uric acid and gastro-intestinal toxæmia have all been invoked. Hurst considers that there may be some relationship to disorder of internal secretion in these women whose attacks occur at the menstrual period and who cease to have migraine after the menopause. He does not regard the other conditions mentioned to be causes at all and considers there is no foundation on which to base these hypotheses. He considers that physical factors may precipitate the onset of migraine in some instances, but the main cause in his opinion is an error of refraction, generally hypermetropic astigmatism, which may or may not be associated with errors in muscle balance due to weakness of the superior and inferior or the external rectus muscles. These errors should be very carefully sought and corrected and repeated tests should be made if relief is not obtained at first. Unilateral headache, nausea and vomiting are the characteristic symptoms, the ocular phenomena being less important. Bilateral headache may be migrainous, but usually it is more pronounced on one side than the other. The author considers that cyclical vomiting in children is closely related to migraine, though not due to eye strain. The main factor in treatment is to prevent eye strain and glasses should be worn continually. "Luminal" and small doses of bromides may be useful until this has been attained. He does not consider that drugs serve any useful purpose during an attack. One reason for this is that digestion appears to be in complete abeyance during the attack. X-ray pictures of the stomach taken during the paroxysm in two patients revealed a complete absence of peristalsis in the stomach and at the end of six hours of voluntary starvation none of the opaque meal had passed into the duodenum. The only means of aborting an attack is by swallowing large quantities of sodium bicarbonate solution (warm) to induce vomiting or by washing out the stomach.

Tests of Liver Function.

O. C. TRAINOR (*Canadian Medical Association Journal*, June, 1924) summarizes the tests used for detection of defective liver function and gives the results of some investigations of the use of phenol-tetrachlor-phthalein as a test of liver function. He states that the known functions of the liver are three: To convert monosaccharides into glycogen and temporarily to store glycogen, to form urea by the synthesis of various protein end products, amino acids, ammonia and so forth and to form bile, partly for the elimination of waste products and partly to aid in the digestion and absorption of fats. He assumes that each individual liver cell may possess in part these threefold functions. If that is so, then any test which reveals a defection in any of the three functions, may be considered as an index of the total functional capacity of the liver. In this way various tests have been tried. The administration of glucose, lævulose or galactose with subsequent tests for glycosuria has been found unreliable. The estimation of the amount of end products of digestion, amino acids, ammonia nitrogen and so forth, which may be expected to be increased when the liver fails to function properly in converting them to urea, has been found impracticable. There is evidence that the fibrinogen in the blood is decreased in severe liver injuries, but as the relationship of liver function to the formation of fibrinogen is not known, this decrease cannot be used as a test. The presence of urobilinogen in the urine is indicative of liver disease. Normally bile pigments are converted in the intestine into urobilinogen which is carried to the liver and reconverted into the ordinary bile pigments. In disease this function of liver cells is impaired and some urobilinogen is excreted unchanged in the urine. The test consists of adding to a few cubic centimetres of urine some crystals of dimethyl-para-amido-benzaldehyde, shaking and rendering acid with hydrochloric acid. An intense red colour indicates a positive reaction. This test yields a reaction in nearly all cases of liver disease, but as it is not quantitative, the test is not ideal. Phenol-tetrachlor-phthalein is said to escape from the body only in the bile and Rowntree endeavoured to detect defects of liver function by estimating the quantity of this dye passed in the fæces following its intravenous administration. This was, however, found rather an unpractical test, being tedious and open to inaccuracies. In 1922 Rosenthal found that after intravenous administration this dye disappeared from the blood rapidly and at a uniform rate in health, but that in disease of the liver due to chloroform or phosphorus poisoning there was definite retention of the dye, with subsequent diminution during convalescence. Five milligrammes of the dye were injected intravenously for every kilogram of body weight. Normally 2% to 6% was present in the blood after fifteen minutes and complete disappearance took place in forty to sixty minutes. In the presence of liver disease the dye may persist in the plasma in high percentages for many hours. Fifteen minutes after the injection blood is withdrawn from a vein in the opposite arm and this is compared colorimetrically with a standard containing ten milligrammes of the dye to one hundred cubic centimetres of distilled water. This standard solution represents 100%, as it is the approximate concentration that would be reached if all the dye were retained. Specimens of plasma (5% sodium hydrate solution being added to bring out the colour) are matched with six dilutions of the above standard solution ranging from 3% to 25% in concentration of dye. The author employed this test in twelve patients, three being healthy. In these the concentration diminished from 5% after twenty minutes to nothing at the end of an hour. In patients with atrophic cirrhosis of the liver, *post partum* eclampsia, carcinoma of the liver, failing heart with arterio-sclerosis, chronic cholecystitis and carcinoma of the head of the pancreas the concentration ranged from 10% to 17% after twenty minutes, to 4% to 15% after sixty minutes. The author considers that this test is an advance on previous methods and advocates its further use to determine its validity.

Goître.

E. A. ARN (*Endocrinology*, May, 1924) endeavours to classify goître and to suggest the best treatment for each type of the condition. He passes over thyreoiditis and malignant disease of the thyreoid as being outside the scope of his discussion and classifies other affections of the thyreoid of a goîtrous nature as colloid goître, adenoma of thyreoid with and without hyperthyreoidism and Graves's disease. Colloid goître usually appears between ten and fourteen years of age, it is smooth and symmetrical and involves both lobes and the isthmus. Sometimes nervous symptoms, tachycardia, tremors and fatigue occur between the ages of sixteen to eighteen; these are generally not due to hyperthyreoidism, since the metabolic rate is normal and the patient usually gains weight. The author discusses prevention of colloid goître by means of drinking boiled water only or administering iodine in the form of sodium iodide added to table salt, giving two grammes of iodine in the spring and autumn, iodine tablets, inhalation of iodine and thyreoid substance by the mouth. Colloid goître may be treated in the same way, the essential being small doses of iodine for a long period. Thyreoid substance is recommended when evidence of impaired thyreoid function appears, such as mental and bodily fatigue, faulty circulation, neuralgia and rheumatic pains. Adenomata usually appear about the age of puberty. They frequently occur as multiple, hard, irregular, movable nodules within the gland. They may cause toxic symptoms following infection or the injudicious use of iodine. Treatment is surgical, but should not be undertaken till after the twenty-fifth year, since the thyreoid is a very essential organ in the years of growth and development. Treatment of adenomata with iodine is strongly condemned. The symptoms of hyperthyreoidism are loss of weight, cardio-vascular changes, gastro-intestinal symptoms, increased metabolism and increased nervous excitability. Graves's disease may

be complicated by enlargement of the thymus gland which is revealed by X-rays. This enlargement can be reduced and the symptoms benefited by X-ray therapy. Two types of Graves's disease are described, one chronic of gradual onset and slow, mild course, the other remittent, characterized by crises during which exacerbations of symptoms occur associated with nausea, vomiting, diarrhœa and extreme debility. If the patient survives, a remission may follow for weeks or months, but recurrence of severe symptoms is common. Recovery may, however, be complete or partial (chronic invalidism without crises). Medical treatment and X-ray treatment are not recommended; the treatment is surgical in the author's opinion, but observation for four to six weeks is advisable, as some patients recover without treatment, while in others the condition proves rapidly fatal in spite of all treatment.

Polio-Myelitis.

F. B. CLARKE (*California and Western Medicine*, September, 1924) reports the results of treatment of polio-myelitis with Rosenow's serum. Flexner and Noguchi isolated a minute organism from the nervous tissue of human beings and monkeys with acute polio-myelitis and by injection of cultures of this organism produced the disease in monkeys. They believed that they had proved the specificity of this organism. Rosenow isolated an organism, a pleomorphic streptococcus, from the central nervous system, mesenteric glands, tonsils and adenoid tissue of patients in different parts of the country. He proved that this organism was absent from various organs in which there were no lesions. He used hundreds of animals to show that this organism had an elective affinity for nervous tissue and that other organisms had no affinity for nervous tissue. He concluded that the organism isolated by himself was the same as that isolated by Flexner and that it varied in size and shape according to the mode of cultivation (aerobic or anaerobic). It was believed that the organism passed directly from the naso-pharynx through the cribriform plate to the pia-arachnoid and adjacent nerve structures. The virus rapidly disappeared from the blood stream when injected, being found in the spleen, bone marrow, lymphatic glands and posterior nerve roots. The virus had been demonstrated in the nasal washings of healthy adults. Anderson and Frost demonstrated the presence of immune bodies in the serum of 66% of persons who had been in close contact with patients without contracting the disease. In some patients paralysis occurs suddenly with little evidence of systemic infection. This mode of onset suggests direct involvement of the nervous system, but when acute general symptoms precede nervous manifestations for some days, this is more suggestive of primary invasion of the blood stream through the tonsils. Clarke considers that the latter is a not infrequent mode of infection. Clarke adopts Wickham's classification into eight types: (i.) Spinal polio-myelitis, (ii.) ascending or Landry's paralysis, (iii.) bublar, (iv.) cerebral, (v.) ataxic, (vi.) polyneuritic, (vii.) meningitic and (viii.) abortive type. The last is important and during an epidemic children who complain of vague symptoms, should be isolated for a week, this being regarded as the incubation period. Meningeal signs are common early in the disease. Rosenow showed that specific antibodies appeared in the serum of horses immunized with aerobic cultures of the pleomorphic streptococcus. Clarke treated eighteen patients with this serum in 1916 with one death, whereas in the rest of the same State in the same year the death rate was 40% in these patients with paralysis, not treated with Rosenow's serum. The dose of serum was five to ten cubic centimetres given intra-muscularly and repeated in twelve to twenty-four hours according to the state of the patient. Rosenow has treated two hundred and fifty-nine patients with this serum with 7.3% mortality and apparent good results as regards paralysis compared with untreated patients.

Hyperglycæmia and Glycosuria in Acromegaly.

ARTHUR W. M. ELLIS (*The Lancet*, June 14, 1924) has reported a case of acromegaly in which the patient manifested both hyperglycæmia and glycosuria. The hyperglycæmia was severe, the blood sugar on admission being estimated at 0.43%. After adequate dieting, a prolonged major operation was successfully accomplished and after operation transient glycosuria only was manifest. The glycosuria and hyperglycæmia ceased to exist after the operation (hypophysectomy). Three years have passed since the patient underwent surgical interference and there has been no return of the glycosuria, although the carbo-hydrate tolerance is diminished. Histological examination of the hypophysis revealed a tumour originating in the *pars intermedia*. The author advances the suggestion that the glycosuria of acromegaly is due to an increase in the activity of the hypophysis, probably the *pars intermedia*, with a resulting interference with the normal action of "Insulin" in carbo-hydrate metabolism.

Latent Syphilis.

J. H. STOKES AND H. A. DES BRISAY (*Canadian Medical Association Journal*, August, 1924) reported the results of investigation at the Mayo Clinic of eight hundred patients suffering with syphilis or suspected syphilitic infection. Seven hundred and fifty-six were examined clinically and by pathological methods (including the Wassermann test of the blood and cerebro-spinal fluid). Careful family and social histories were taken in these patients to determine a cause for their symptoms. In none of them was a definite diagnosis possible by ordinary methods of examination and yet 60% yielded positive results enabling the authors to make a diagnosis of syphilis. Of two hundred and fifty-five patients with syphilis 41% were unaware that they had been infected with any venereal disease and 52% did not know they had acquired syphilis. Only 27% gave a history of a genital sore and only 10% gave histories of secondary symptoms. It was noticed that whereas only 23% of women suffering with unrecognized syphilis could give any historical detail that would aid to diagnosis, 81% of men could recall some contributing facts. The authors lay stress on this evidence of the masked or concealed onset of syphilis and emphasize the importance of glandular swellings as an aid to diagnosis. Of two hundred and eight patients suffering from syphilis only 68% yielded Wassermann reactions. The authors consider that the Wassermann test has a 30% margin of error. Of forty-six patients who had received treatment for syphilis at an earlier date, 60% gave a reaction to the Wassermann test. These figures suggest that treatment by the mouth does not modify to any extent the serological findings of later years. The authors point out that neuro-syphilis exists with failure to produce a Wassermann reaction in the blood much more frequently than any other syphilitic affection. In only 41% of neuro-syphilitic patients was the result of the Wassermann test positive, whereas in other forms, visceral, cardio-vascular, osseous, cutaneous and mucous membrane infection, 80% or more positive results were obtained. Again, of sixty patients whose blood did not yield a reaction to the Wassermann test, 62% had abnormal spinal fluid. Of ninety patients with a reaction to the Wassermann test, only 40% had abnormal cerebro-spinal fluid.

Vaquez's Disease.

L. BELTZ AND E. KAUFMANN (*Klinische Wochenschrift*, October, 1924) give the history of a case of Vaquez's disease (*polycythæmia rubra*). The total bulk of the blood was enormously increased, while the ratio of plasma to red corpuscles was considerably lowered. Owing to this condensation of plasma, the albumin percentage was raised. In the treatment venesection followed by saline injections is indicated, especially when there is a tendency to thrombosis.

Pathogenesis of Diabetes Insipidus.

E. MEYER AND R. MEYER-BISCH (*Klinische Wochenschrift*, September 30, 1924) give the history of a case of *diabetes insipidus* observed from the commencement of the illness to the termination. The condition was found to be due to a sarcoma involving the posterior lobe and infundibulum of the pituitary body, as well as a large part of the mid-brain. The temperature was raised, hyperglycæmia was present and the sodium chloride content of the blood lowered. There was a primary lowering of the renal concentration without any disturbance of kidney tissue. Administration of "Pituglandol" was unsuccessful.

British Medical Association News.

MEDICO-POLITICAL.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Walter and Eliza Hall Institute, Melbourne Hospital, on February 11, 1925, DR. STANLEY ARGYLE, the President, in the chair.

Fellowship of Medicine and Post-Graduate Medical Association.

THE SECRETARY read a letter which had recently been received by the Council from Dr. M. Roy, Secretary of the Fellowship of Medicine and Post-Graduate Medical Association, 1, Wimpole Street, London, W.1. The letter was published in THE MEDICAL JOURNAL OF AUSTRALIA, February 28, 1925. Copies of the syllabuses of post-graduate courses in England were enclosed and the file was laid on the table for the perusal of members interested.

Presentation of Balance Sheets.

The duly audited balance sheets of the Victorian Branch of the British Medical Association, of the Medical Society of Victoria and of the Medical Agency were summarized and presented for adoption by Dr. C. H. Mollison, the Honorary Treasurer. Members were provided with copies of the balance sheets, published herewith.

In moving the adoption of the balance sheets as presented by Dr. Mollison, DR. A. L. KENNY said that he wished to give expression to a feeling which was general among the members of the Branch, the sense of great indebtedness to Dr. Mollison for his faithful service in his arduous office. All would echo the hope that Dr. Mollison would be spared for many years and that he would long continue to act as Honorary Treasurer of the Branch. The motion was seconded by Dr. A. V. M. Anderson and carried with acclamation.

Dr. Mollison briefly expressed his thanks.

Notice of Motion.

DR. J. W. DUNBAR HOOPER had given notice of the following motion:

> This Branch does not consider that the present system of maternity allowances is the best way of reducing maternal mortality and morbidity and infantile mortality.

Dr. Hooper forwarded an apology for his unavoidable absence and asked that the foregoing motion be placed on the notice paper for the next meeting of the Branch. On the motion of Dr. J. F. Wilkinson, seconded by Dr. F. L. Davies, it was agreed that the motion be deferred as requested by Dr. Dunbar Hooper.

Medicine in Other Countries.

DR. J. F. MACKEDDIE delivered an address on impressions of medicine abroad as gathered on a recent visit to England and America.

In an account of the medical work in progress at the various centres which he had visited, Dr. Mackeddie discussed developments in research regarding asthma and hay fever, peptic ulcer and the specific serum therapy

VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

Statement of Revenue and Expenditure for Period from January 23, 1924, to January 15, 1925.

EXPENDITURE.

	£	s.	d.
To Medical Society of Victoria	1,784	4	9
„ British Medical Association	1,360	0	3
„ Federal Committee	109	10	0
„ Assistant Secretary	177	14	0
„ Postages, Duty Stamps, etc.	59	14	6
„ Printing	94	17	9
„ Stationery	44	5	1
„ Rebates to Divisions	50	6	6
„ Transfer to Building Fund	800	0	0
„ Refund to Medical Agency *re* Delegate, London	114	2	2
„ Commission on New Members	9	0	0
„ Advertising	10	3	0
„ Audit, 1923	3	3	0
„ Telephone	7	5	6
„ Clerical Assistance	16	12	0
„ Electric Light	7	5	5
„ Part Cost of New Typewriter	10	4	0
„ Travelling Expenses	4	13	5
„ Obstetric Committee Expenses	19	19	0
„ War Memorial	10	0	0
„ Donation, Melbourne Hospital	10	10	0
„ Bonus, Assistant Melbourne Hospital	5	0	0
„ Hire of Playhouse	11	2	4
„ Hire of Halls for Divisional Meetings	2	1	0
„ Expenses *re* Unveiling of Portrait, Sir George Syme	17	6	6
„ Sundries	8	19	3
„ Balance—			
Cash in Bank of Victoria, Ltd., Prahran — £183 11 0			
Organization Fund — 100 0 0			
Cash in Hand — 12 0 0			
	295	11	0
	£5,043	10	5

REVENUE.

	£	s.	d.
By Balance, January 23, 1924	997	0	6
„ Subscriptions—			
Metropolitan Members — £2,319 11 3			
Country Members — 1,257 11 6			
Junior Members — 441 9 6			
	4,018	12	3
„ Bank Discount	24	9	2
„ Congress Ball Balance	2	15	0
„ Sale of Journals		13	6
	£5,043	10	5

Audited and found correct—
J. V. M. WOOD & CO., PTY., LTD.,
Per ERNEST G. MARKS,
Incorporated Accountants and Auditors.
Melbourne, Second Day of February, 1925.

C. STANTON CROUCH,
Secretary.
C. H. MOLLISON,
Honorary Treasurer.

MEDICAL SOCIETY OF VICTORIA.

Statement of Revenue and Expenditure for Period from January 23, 1924, to January 15, 1925.

Expenditure.	£	s.	d.
To The Medical Journal of Australia	1,050	10	0
„ Salaries—			
Secretary £377 13 4			
Assistant Secretary (bonus) 5 0 0			
Library Clerk 52 13 4			
Caretaker 52 13 4			
	488	0	0
„ Library, Journals and Binding	28	0	1
„ Part Cost New Typewriter	10	0	0
„ Postages	69	17	7
„ Rates, Taxes and Insurance	18	6	8
„ Light and Power	7	5	5
„ Audit for 1922	4	4	0
„ Bank Charges		5	0
„ Repairs	4	11	3
„ Telephone	7	5	8
„ University Research Fund	26	5	0
„ Balance, Bank of Victoria, Ltd., Prahran	304	13	10
	£2,019	4	6

Revenue.	£	s.	d.
By Balance, January 23, 1924	213	19	9
„ Subscriptions	1,784	4	9
„ Sale of Two Examination Couches	21	0	0
	£2,019	4	6

Audited and found correct—
J. V. M. WOOD & CO., PTY., LTD.,
Per ERNEST G. MARKS,
Incorporated Accountants and Auditors.

Melbourne, Second Day of February, 1925.

C. STANTON CROUCH,
Secretary.
C. H. MOLLISON,
Honorary Treasurer.

of lobar pneumonia. He indicated that the refinement in the preparation of anti-pneumococcal serum by which the globulin fraction was removed from the serum without impairment of its anti-body content, had been successfully accomplished and he emphasized the importance of this advance in minimizing the risk of severe reactions after the intravenous injection of the serum.

The major portion of Dr. Mackeddie's address consisted of an entertaining account of his studies in the clinics of eminent London neurologists.

By means of lantern slides he effectively demonstrated the use of intrathecal injections of "Lipiodol" combined with skiagraphy in the localization of pathological processes in the spinal cord. "Lipiodol," a 40% solution of iodine in oil, was introduced into the spinal theca *viâ* the *cisterna magna* or by the route commonly used for intrathecal injections after lumbar puncture and sometimes by both paths. The existence of obstruction to the free circulation of cerebro-spinal fluid in the sub-arachnoid space and the point at which such obstruction occurred, was disclosed in the skiagram taken after the injection of "Lipiodol" in much the same manner as irregularities in the urinary tract were demonstrated by a pyelogram. No bad effects attended the introduction of "Lipiodol" to the theca, although the heavy solution was very slow of absorption and remained in the spinal canal for many months. The method was largely employed by Purves Stewart and by Sargent at Westminster.

A cinematograph film was displayed in which the technique of the withdrawal of cerebro-spinal fluid from the *cisterna magna* and the introduction of "Lipiodol" to the sub-arachnoid space by this route was clearly demonstrated.

Another film recorded the clinical examination by a prominent neurologist of a patient in whom suppuration had occasioned extensive destructive changes in the left lobe of the cerebellum. The objective signs in Benedict's syndrome were well illustrated in a third motion picture.

Dr. Mackeddie also dealt briefly with the diagnostic method described as ventriculography, in which radiograms are taken after the injection of air into the cerebral ventricles with the object of determining the existence of undue distension or of distortion of their outlines.

Procedure in Passing Resolutions as to Professional Conduct.

Dr. L. S. Latham moved that consideration of the rules governing the procedure in passing resolutions as to professional conduct be placed on the notice paper for the next meeting of the Branch. The motion was seconded by Dr. H. Douglas Stephens and carried unanimously.

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A meeting of the Medical Sciences Club of South Australia was held at the University of Adelaide on October 3, 1924.

Structure and Action of Plain Muscle.

Dr. O. W. Tiegs gave an account of the structure and action of plain muscle. From the fact that the myofibrillæ were thicker in contracted regions of the cell he inferred that they were not artefacts as was often supposed, but were actively concerned in plain muscle contraction. Their intimate structure was probably similar to that of the myofibrillæ of skeletal and cardiac muscle, the muscle machine consisting of a collapsible sac opening by a capillary tube into a porous tissue, the whole on an ultramicroscopical scale. Contraction was evidently due to liberation of lactic acid on to the walls of the myofibrillæ, this giving rise to movements of the fluid contents of these collapsible sacs, under the influence of the surface tension changes which result.

Relaxation from the contracted state was due to the liberation of an unknown basic substance, exactly as in skeletal muscle. Tonus was evidently due to the liberation of the acid alone in a certain number of fibres (depending, of course, upon the degree of the tonus) and inhibition from the tonically contracted state would be the result of the liberation of the basic substance into these contracted fibres.

In conformity with this view he found that plain muscle fibres had a double innervation.

From the fact that the chemical reaction which produced contraction, occurred in the central nucleated region of the fibre, it was shown how the shape of the ordinary "twitch" curve could be deduced.

He also pointed out that there was no evidence that conduction of stimuli occurred within the fibres. There could, therefore, be none from fibre to fibre and hence the conception of myogenic plain muscle movement must be fallacious.

Botulism.

DR. L. B. BULL stated that so far as was known no case of botulism in the human subject had been reported in Australia. In horses and cows, however, botulism was known to occur and the possibility of its occurrence in the human subject should not be lost sight of. In Europe and America cases had occurred mainly associated with the ingestion of canned vegetable products. These cases had been due to the ingestion of preformed toxin in spoiled foods contaminated with *Bacillus botulinus*, Types A and B. Up to the present neither of these types had been found in Australia, but Type C had been isolated by Seddon from a decomposed carcase of a cow and Dr. Buli had demonstrated its presence in forage. Recently also he had been able to demonstrate the presence in soil of another member of this group. At least, as far as experiments had gone, he had been unable to prove that it was either Type A, B or C. In the treatment of botulism with antitoxin it was necessary to use one prepared from the type producing the symptoms. Antitoxins, Types A and B, were in use in Europe and America, but the evidence so far would indicate that these antitoxins might be useless in the treatment of cases in Australia. This was another illustration of the fact that Australians had their own problems to solve and could not rest entirely upon the work done in other countries.

As work progressed they would probably find that *Bacillus botulinus* contained a fairly big group, the individual types possessing an homologous toxin.

Bacillus botulinus was a ubiquitous organism. Meyer and co-workers had shown that on examination of 624 specimens of soil, vegetables, fruits, feeds, manure and sewage collected in 36 counties of California, 30% of the samples produced toxic cultures.

A further study in 1,538 soil, vegetable, feed and manure specimens of every State in the United States of America except Virginia, showed that *Bacillus botulinus* was a common soil anaerobe of the Western States of the Cordilleran system. It was less frequently encountered in Atlantic States and was relatively rare in Middle States, the great plains and Mississippi valley. *Bacillus botulinus* spores were far more prevalent in virgin and pasture soils than in dirt, soil or manure collected from animal corrals, pig pens and so forth.

In Alaska and Canada *Bacillus botulinus* was not found in the coast land soil of the Aleutian Archipelago. It was found in moraine, glacier and mountain soil around Lake Louise in the Canadian Rockies, also from provinces of Prince Edward Island, Nova Scotia, Quebec, Ontario and British Columbia.

Bacillus botulinus, Type B, had been demonstrated in soil and vegetable specimens collected in Belgium, Denmark, England, the Netherlands and Switzerland. Type A had been consistently absent.

For many years it was believed that the toxin of *Bacillus botulinus* produced symptoms by its action on the cells of the central nervous system. Recent work had gone to show that the old conception was probably wrong. Semerak (1921) had claimed that the changes produced by the toxin were confined to the vascular system. Thrombosis in arteries and veins was the initial change, followed by ischemic necrosis and later by inflammation. It had no direct action on nerve cells; the retrogressive changes were secondary and due to the disturbed blood supply.

Cowdy and Nicholson (1924) had examined the tissues of mice, guinea pigs and rabbits which had been subjected to toxin and had been unable to observe any evidence of the action of toxin on the central nervous system. No changes were found in Nissl's bodies or nuclei of the nerve cells. Nerve fibres were found to show no microscopical evidence of injury. The vascular changes were slight and infrequent. Their observations tended to show that upon the histological side there was no evidence inconsistent with the results of physiological experiments indicating that the site of the action of the toxin was upon peripheral nerve terminals. Most important observations on the action of the toxin had been made by Dickson and Shevky in 1923. They showed that the action of the toxin upon the nervous system was peripheral and not central. It affected the peripheral endings of the motor fibres of portions of the autonomic system and of the voluntary nervous system. They had not ascertained whether damage was located in terminal nerve endings or in the myo-neural junction and the type of damage was unknown save that it was not of the nature of an organic destruction of tissue. There was no demonstrable change in muscle cell, striated or smooth. The effect upon the autonomic system was confined to the motor and secretory fibres which constituted the para-sympathetic system and resulted in a relatively unstable blocking of the nerve impulses. This was manifested by disturbed function, but not true paralysis of the muscles which they supplied. The fibres of the sympathetic system were not affected. The effect upon the voluntary nervous system was less severe. There was a rapid and progressive fatigue. The sensory fibres of the peripheral nerves and the reflex areas were not affected. The nature of the interference with conduction of nerve impulses had not been ascertained.

Obituary.

FRIEDRICH TRENDELENBURG.

THE death of Professor Friedrich Trendelenburg has been announced. Trendelenburg was eighty years of age. He enjoyed a great international reputation as a surgeon. His father was a well-known philosopher. Friedrich Trendelenburg was a student of medicine at Berlin, Edinburgh and Glasgow. He became assistant to von Langenbeck in Berlin in 1868 and in 1874 he was appointed Professor of Surgery at Rostock. In 1882 he was transferred to Bonn, where he remained for thirteen years. In 1895 he accepted the chair of surgery at Leipzig. Apart from his brilliance as a teacher of surgery, Trendelenburg earned renown as an original thinker and reformer. Several operations devised by him have found their way into surgical practice, while many instruments planned by him are in general use. The Trendelenburg position is known to students of medicine throughout the world. He had a wonderful memory and never forgot the name of a patient or old student. He was an eloquent speaker and a good linguist.

WILLIAM POPE SEED.

WE regret to announce the death of Dr. William Pope Seed, which occurred at Perth, Western Australia, on February 21, 1925.

EUSTACE JULIAN KEOGH.

WE regret to announce the death of Dr. Eustace Julian Keogh, of South Yarra, Victoria.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING OF THE SENATE OF THE UNIVERSITY OF SYDNEY was held on February 2, 1925.

The degree of master of surgery was conferred *in absentia* on Drs. T. M. S. Hall and J. G. Stephens.

Letters were received from the Chancellor of the University of Western Australia, the Speaker of the Legislative Assembly of New South Wales, the Brisbane Hospital Clinical Society and the Board of Directors of the

Royal North Shore Hospital with expressions of sympathy at the death of Professor Hunter.

A cablegram was received from Professor MacCallum, accepting the position of Vice-Chancellor for the current year. Professor MacCallum is at present in England, where he is to deliver a lecture this month before the members of the British Academy. He is to receive the honorary degree of Litt.D. from the University of Oxford.

The following resolution was passed by the Senate in regard to the death of Professor Haswell:

> The Senate records with sincere regret the death of Professor W. A. Haswell, Emeritus Professor of Zoology, which occured on January 24. Professor Haswell's eminence as a scholar was acknowledged by the whole scientific world and his gentle, courteous nature endeared him to all his students. He rendered eminent service to the University and to the cause of scientific education by his great gifts as a scholar and a teacher. Deeply interested in the problems of biological study, he never failed to illumine them from his stores of knowledge of the past and he has left valuable contributions to the subject of which he was an acknowledged master.

A gift of the late Professor Haswell's microscope slides, manuscripts and drawings, a microtome and other apparatus for section cutting, together with three cabinets for slides and specimens was received from Mrs. Haswell. It was decided to send a letter of grateful thanks to the donor.

The following appointments and re-appointments were made to the teaching staff:

Drs. W. N. Horsfall and W. W. Ingram as Demonstrators in Physiology.
Miss Lucy Wood, B.A., Demonstrator in Zoology.
Dr. W. A. Ramsay Sharp, Tutor in Surgery at Sydney Hospital.
Demonstrator in Physics: Re-appointment of Mr. I. S. Turner, B.Sc..
Dr. G. H. Abbott, Lecturer in Clinical Surgery.
Dr. Gordon Craig, Lecturer in Clinical Surgery.
Dr. St. J. W. Dansey, Lecturer in Clinical Surgery.
Dr. C. B. Blackburn, Lecturer in Clinical Medicine.
Dr. Sinclair Gillies, Lecturer in Clinical Medicine.
Dr. E. W. Fairfax, Lecturer in Clinical Medicine.
Dr. H. J. Clayton, Tutor in Medicine, Royal Prince Alfred Hospital.
Dr. C. G. McDonald, Tutor in Medicine, Royal Prince Alfred Hospital.
Dr. M. C. Lidwill, Tutor in Anæsthetics, Royal Prince Alfred Hospital.
Dr. J. M. Gill, Lecturer in Clinical Medicine, Sydney Hospital.
Dr. C. E. Corlette, Lecturer in Clinical Surgery, Sydney Hospital.
Dr. G. C. Willcocks, Tutor in Medicine, Sydney Hospital.
Dr. B. T. Edye, Demonstrator in Pathology.
Dr. Marjory Little, Demonstrator in Pathology.

Dr. A. W. Holmes à Court was appointed as co-examiner with Dr. J. MacPherson in Therapeutics and *Materia Medica*.

The following were appointed to represent the University of Sydney at the forthcoming conference of the Universities of the British Empire at London and Cambridge in July next: The Chancellor (the Hon. Sir William Cullen), Professor Sir Edgeworth David, Professor J. T. Wilson and Sir Josiah Symon.

Correspondence.

ROUTINE TREATMENT FOR TYPHOID.

SIR: Very many practitioners who were brought up on the "milk and water" doctrine as regards the dietetics of enteric fever will welcome heartily the confirmation of their change of views which is afforded by Dr. Kinsella's paper published February 21.

But the stolidity of routine applies not only to the diet of typhoid patients. The recumbent posture and the routine hydrotherapy have become more articles of faith than matters of scientific belief.

Since 1912 my practice has been to advise every patient with enteric to sit up in bed or out of bed as may be most conveniently managed, as often and as long as he or she feels inclined and able to do so. The milder cases are encouraged to walk about when their condition warrants it.

This was the routine for about twelve months at the Brisbane Hospital when I had charge of the enterics. During that time there was, I think, one death and there was no thrombosis in any case. I have not had time to analyse the figures as to time in hospital and in any case the numbers would be too small to carry weight as statistics; but I am confident from my experience of hospital and of private patients that the duration of the illness is shorter and the convalescence more rapid. In hospital the immersion bath was discarded and the frequency of the sponging was greatly reduced. Except in the one fatal case abdominal distension was practically unknown.

Since 1919 I have seen but few cases of enteric and am out of touch with recent literature, but as the melting pot is evidently still hot, I venture to suggest that these ideas might also be worthy of a place therein.

Yours, etc.,

C. A. THELANDER.

Brisbane, February 26, 1925.

WHOOPING COUGH TREATED BY RECTAL ADMINISTRATION OF ETHER.

SIR: Ether has long been known as a remedy for this disease, but when given *per os* its flavour and liability to be vomited and the pain of hypodermic injection hinder its use by these routes. Since general anæsthesia can be produced by ether in oil *per rectum*, it occurred to me to use this method and four cubic centimetres of ether in sixteen cubic centimetres of olive oil was the first dose tried, the patient being a man of fifty-five years. Relief was prompt and he secured his first sleep for nearly a week other than short snatches. After a good night, slight infrequent attacks during the next day, a second dose was indicated at night and again the next night, after that on alternate nights a few times; since when it has not been required. Another man also suffering very severely experienced great relief from the first dose, the subsequent course of the disease being completely altered. All children have been greatly relieved except those of fifteen months and under who failed to retain the injection.

Yours, etc.,

C. JOYCE.

Kelmscott, Western Australia,
February 9, 1925.

DIPLOPIA IN DREAMS.

SIR: In the waking state it universally holds that any object will appear double, if the eyes are accommodated for a point much nearer or much further than the object and in or nearly in the same line of vision. In dreams this is not or need not be the case. I had a vivid visual dream last night, whose genesis could be clearly analysed, in which I was attending a patient in whom the question of diplopia arose. I incidentally mentioned the above physiological diplopia and got the patient and his wife to go through simple tests of its truth and carefully and vividly went through test after test myself. But in no single instance could I see the primary object double. I was highly nettled. As soon as I awoke, I tested myself and found things normal.

As sharply-cut visual images play such an immense part in dreams, the above limitation to the dream's power seems to me to be both interesting and significant.

Have any of your readers had or heard of any cognate experience?

Yours, etc.,

W. OSTERMEYER, M.A., M.D..

Carlton, Melbourne,
February 13, 1925.

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered under the provisions of the *Medical Act of 1867* as duly qualified medical practitioners:

Horn, Harold W., M.B., Ch.M., 1923 (Univ. Sydney), East Brisbane.

Gibson, W. Lockhart, M.B., Ch.M., 1924 (Univ. Sydney), Brisbane.

Cole, Colin Richard, M.B., Ch.M., 1924 (Univ. Sydney), Milmerran.

NEW SOUTH WALES.

THE following have been been registered under the provisions of the *Medical Act, 1912 and 1915*, as duly qualified medical practitioners:

Delépine, Jules, M.B., Ch.M., 1885 (Univ. Edinburgh).

Mackenzie, Ian, M.B., Ch.B., 1916 (Univ. Edinburgh), No. 1, Glenayr Avenue, Bondi.

Additional Qualifications.

Woodhill, Victor Richmond, M.R.C.P. (London).

Books Received.

THE PRACTICAL MEDICINE SERIES, COMPRISING EIGHT VOLUMES ON THE YEAR'S PROGRESS IN MEDICINE AND SURGERY: Under the General Editorial Charge of Charles L. Mix, A.M., M.D.: Volume II.: General Surgery; 1924. Chicago: The Year Book Publishers. Crown 8vo., pp. 706, with illustrations.

LORD LISTER, by Sir Rickman John Godlee, Bt., K.C.V.O., M.S., F.R.C.S.; Third Edition Revised; 1924. Oxford: The Clarendon Press. Royal 8vo., pp. xvi. + 685.

A SYNOPSIS OF SPECIAL SUBJECTS FOR THE USE OF PRACTITIONERS: Diseases of the Skin, by Henry C. Semon, M.A., M.D. (Oxon.), M.R.C.P. (London); Obstetrics and Diseases of Women, by Malcolm Donaldson, M.B., B.Ch. (Cantab.), F.R.C.S. (England); The Ear, Nose and Throat, by Archer Ryland, F.R.C.S. (Edinburgh); The Eye, by J. F. Cunningham, O.B.E., F.R.C.S. (England); 1924. London: H. K. Lewis & Company, Limited. Demy 8vo., pp. viii. + 373. Price: 18s. net.

Medical Appointments.

Dr. Henry Rogerson (B.M.A.) has been appointed acting Medical Superintendent of the Hospital for the Insane at Kew, Victoria.

* * * *

Dr. Walter Lockhart Gibson (B.M.A.) has been appointed Assistant Resident Medical Officer at the General Hospital, Ipswich, Queensland.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xiv.

AUSTIN HOSPITAL, HEIDELBERG, VICTORIA: (a) Senior Resident Medical Officer, (b) Honorary Dermatologist.

BURREN JUNCTION, NEW SOUTH WALES: Medical Practitioner.

DEPARTMENT OF DEFENCE: Director-General of Medical Services.

DEPARTMENT OF PUBLIC INSTRUCTION, MELBOURNE: Four Vacancies.

LAUNCESTON PUBLIC HOSPITAL, TASMANIA: Two Junior Medical Officers.

QUEEN VICTORIA HOSPITAL, MELBOURNE: Medical Superintendent.

RENWICK HOSPITAL, OUT-PATIENTS' DEPARTMENT, SYDNEY: Honorary Physician.

ROYAL AUSTRALIAN NAVY: Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C..

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited Mutual National Provident Club. National Provident Association.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital.
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Diary for the Month.

MAR. 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAR. 10.—Tasmanian Branch, B.M.A.: Branch.
MAR. 12.—Victorian Branch, B.M.A.: Council.
MAR. 12.—South Australian Branch, B.M.A.: Council.
MAR. 13.—Queensland Branch, B.M.A.: Council.
MAR. 17.—New South Wales Branch, B.M.A.: Medical Politics Committee: Organization and Science Committee.
MAR. 17.—Tasmanian Branch, B.M.A.: Council.
MAR. 18.—Western Australian Branch, B.M.A.: Branch.
MAR. 18.—Western Medical Association, New South Wales.
MAR. 24.—New South Wales Branch, B.M.A.: Council (Quarterly).
MAR. 25.—Victorian Branch, B.M.A.: Council.
MAR. 26.—New South Wales Branch, B.M.A.: Branch (Annual).
MAR. 27.—Queensland Branch, B.M.A.: Council.
MAR. 31.—New South Wales Branch, B.M.A.: Council.
MAR. 31.—Section of Hygiene and Preventive Medicine, New South Wales Branch, B.M.A.
APR. 1.—Victorian Branch, B.M.A.: Branch.
APR. 7.—Tasmanian Branch, B.M.A.: Council.
APR. 9.—New South Wales Branch, B.M.A.: Clinical Meeting.
APR. 9.—Victorian Branch, B.M.A.: Council.
APR. 14.—New South Wales Branch, B.M.A.: Ethics Committee.
APR. 14.—Tasmanian Branch, B.M.A.: Branch.

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